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Macmillan's Manuals of
Medicine and Surgery

A MANUAL OF MEDICINE



A

Manual of Medicine

EDITED BY

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ROYAL COLLEGE OF PHYSICIANS OF LONDON, AND FOR THE
BRITISH AND INDIAN ARMY MEDICAL SERVICES

VOL. IV.

DISEASES OF THE RESPIRATORY AND OF THE
CIRCULATORY SYSTEMS

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CONTENTS

	PAGE
CONTRIBUTORS	xi
<i>THE GENERAL ANATOMY AND PHYSIOLOGY OF THE RESPIRATORY</i>	
<i>SYSTEM</i>	I
DISEASES OF THE RESPIRATORY SYSTEM	25
<i>DISORDERS OF THE UPPER RESPIRATORY TRACT</i>	25
Acute Nasal Catarrh	25
Epistaxis	29
Adenoids	33
Hay Fever	40
Some Special Affections of the Nasal Mucous Membrane	46
<i>DISEASES OF THE LOWER RESPIRATORY TRACT</i>	50
GENERAL ETIOLOGY	50
MORBID ANATOMY AND GENERAL PATHOLOGY	60
GENERAL SYMPTOMATOLOGY	101
PHYSICAL EXAMINATION OF THE LUNGS	123
<i>DISEASES OF THE LARYNX</i>	139
ACUTE CATARRHAL LARYNGITIS	139
ŒDEMA OF THE LARYNX	140
CHRONIC LARYNGITIS	141
PERICHONDritis OF THE LARYNX	143
DISEASE OF CRICO-ARYTENOID JOINT—LARYNGEAL STENOSIS—	
SYPHILIS OF THE LARYNX	144
TUBERCULOSIS OF THE LARYNX	145
LARYNGEAL GROWTHS	148
LARYNGEAL NEUROSES	151
<i>DISEASES OF THE TRACHEA AND BRONCHII</i>	155
ACUTE TRACHEITIS AND BRONCHITIS	155
CAPILLARY BRONCHITIS	161
CHRONIC BRONCHITIS	162
PLASTIC AND FIBRINOUS BRONCHITIS	167

	PAGE
BRONCHIECTASIS	170
STENOSIS OF THE TRACHEA AND BRONCHI	173
FOREIGN BODIES IN THE AIR PASSAGES	174
ASTHMA	175
<i>DISEASES OF THE LUNGS</i>	183
PULMONARY EMPHYSEMA	183
PULMONARY CONGESTION AND ŒDEMA	186
PULMONARY EMBOLISM AND THROMBOSIS	188
BRONCHO-PNEUMONIA	190
PULMONARY COLLAPSE	195
ACUTE PNEUMONIA	196
PULMONARY ABSCESS	209
PULMONARY GANGRENE	210
CHRONIC PNEUMONIA	212
PULMONARY TUBERCULOSIS — ACUTE MILIARY PULMONARY	
TUBERCULOSIS	214
ACUTE PNEUMONIC PHTHISIS	217
CHRONIC PULMONARY TUBERCULOSIS	219
PULMONARY GROWTHS	243
HYDATIDS OF THE LUNG	246
SYPHILIS OF TRACHEA, BRONCHI, AND LUNGS	248
<i>DISEASES OF THE PLEURA</i>	250
GENERAL ETIOLOGY	250
MORBID ANATOMY AND PATHOLOGY	252
PLEURISY—ACUTE	256
EMPYEMA	259
CHRONIC PLEURISY	260
PNEUMOTHORAX—PYO-PNEUMOTHORAX	268
TUBERCULOSIS OF THE PLEURA—MORBID GROWTHS IN PLEURA	272
HYDATIDS OF THE PLEURA:	273
<i>DISEASES OF THE MEDIASTINUM</i>	274
MEDIASTITIS	274
DISEASES OF THE MEDIASTINAL LYMPHATIC GLANDS	277
NEW GROWTHS OF THE MEDIASTINUM	277
<i>DISORDERS OF THE DIAPHRAGM</i>	283
<i>GENERAL ANATOMY AND PHYSIOLOGY OF THE VASCULAR SYSTEM</i>	288
DISEASES OF THE CIRCULATORY SYSTEM	317
<i>DISEASES OF THE HEART</i> —GENERAL ETIOLOGY	317
GENERAL MORBID ANATOMY AND PATHOLOGY	323

CONTENTS

vii

	PAGE
PHYSICAL EXAMINATION OF THE HEART AND VESSELS	339
GENERAL SYMPTOMATOLOGY	360
GENERAL COURSE AND PROGNOSIS	376
DISEASES OF THE ENDOCARDIUM	396
ACUTE ENDOCARDITIS	396
CHRONIC VALVULAR DISEASE	398
DISEASES OF THE MYOCARDIUM	415
ACUTE MYOCARDITIS—CHRONIC MYOCARDITIS	415
CARDIAC ANEURYSM—FATTY DEGENERATION AND INFILTRA- TION	417
CARDIAC SYPHILIS	418
ENLARGEMENT OF THE HEART, DILATATION, AND HYPER- TROPHY	419
Rarer MYOCARDIAL LESIONS—RUPTURE—WOUNDS	422
NEW GROWTHS—PARASITES	422
CARDIAC STRAIN	423
DISEASES OF THE PERICARDIUM	425
ACUTE PERICARDITIS	425
CHRONIC PERICARDITIS—ADHERENT PERICARDIUM	427
TUBERCULOUS PERICARDITIS	429
HYDROPERICARDIUM—HÆMOPERICARDIUM—PYO-PNEUMO-PERI- CARDITIS	429
FUNCTIONAL DISORDERS	430
CONGENITAL AFFECTIONS	433
<i>DISEASES OF THE BLOOD-VESSELS</i>	438
DISEASES OF ARTERIES	438
GENERAL ETIOLOGY	438
GENERAL MORBID ANATOMY AND PATHOLOGY	439
ARTERITIS, ACUTE AND CHRONIC	443
EMBOLISM	447
ATHEROMA	448
ANEURYSM—THORACIC	450
DISEASES OF THE PULMONARY ARTERY	457
DISEASES OF VEINS	460
CEDEMA AND DROPSY	464
INDEX	489

ILLUSTRATIONS

FIG.	PAGE
1. Diagrammatic Representation of the Actions of the Laryngeal Muscles	2
2. Normal Respiratory Curve showing Time of Associated Sounds .	13
3. Section of Bronchial Mucosa showing Acute Bronchitis (Ziegler) .	63
4. Pulmonary Alveolus showing Broncho-pneumonia (Ziegler) . .	65
5. Section of Lung showing Broncho-pneumonia (Ziegler) . .	66
6. Acute Lobar Pneumonia, Red Hepatisation (Hamilton) . .	67
7. Acute Lobar Pneumonia, Gray Hepatisation (Hamilton) . .	68
8. Pulmonary Capillaries and Epithelium in Passive Congestion (Hamilton)	75
9. Chronic Hypertrophous Emphysema (Hamilton) . . .	83
10. Miliary Tuberculous Broncho-pneumonia (Ziegler) . . .	92
11. Chronic Pulmonary Tuberculosis with Thickened Pleura . .	94
12, 13, 14, and 15. Areas of Reflected Visceral Pain and Tenderness in Cardiac and Pulmonary Diseases	120 and 121
16. Diagram of Distribution of Velocity and Pressure in the Vascular System	290
17. Diagram of Intracardiac and Aortic Pressure, Time of Sounds, and Impulse	300
18 and 19. Normal Pulse Curves	303
20 and 21. Dicrotic and Anacrotic Pulse Curves	303
22. Fibroid Disease of the Heart (Ziegler)	325
23. Chronic Valvular Disease of Aortic Valves (Ziegler)	328
24. Chronic Valvular Disease of Mitral Valve (Ziegler)	328
25. Acute Endocarditis of an Aortic Cusp (Ziegler)	329
26. Acute Pericarditis (Ziegler)	330
27. Hypertrophy of Left Ventricle (Ziegler)	332
28. Rupture of the Heart (Ziegler)	334
29. Comparative Pulse Curves	354

FIG.		PAGE
30.	Arteritis obliterans (Hamilton)	441
31.	Syphilitic Arteritis (Ziegler)	442
32.	Atheroma (Hamilton)	443

COLOURED PLATES

I. and II.	Diagrammatic Representation of the Position of the Thoracic Viscera	<i>Between</i> pp. xii and i
III.	Schema of the Circulation	<i>Facing</i> p. 306

TABLE

Showing the Levels of various Structures in the Thorax in relation to the Spines and Bodies of the Vertebrae	<i>Between</i> pp. xii and i
---	------------------------------

CHARTS

Respiration Curves in Dyspnoea	114
Temperature Curve in Broncho-pneumonia	192
Temperature Curve in Pneumonia	198
Temperature Curve in Miliary Pulmonary Tuberculosis	216
Temperature Curve in Caseating Pulmonary Tuberculosis	222
Temperature Curve showing Tuberculin Reaction	230

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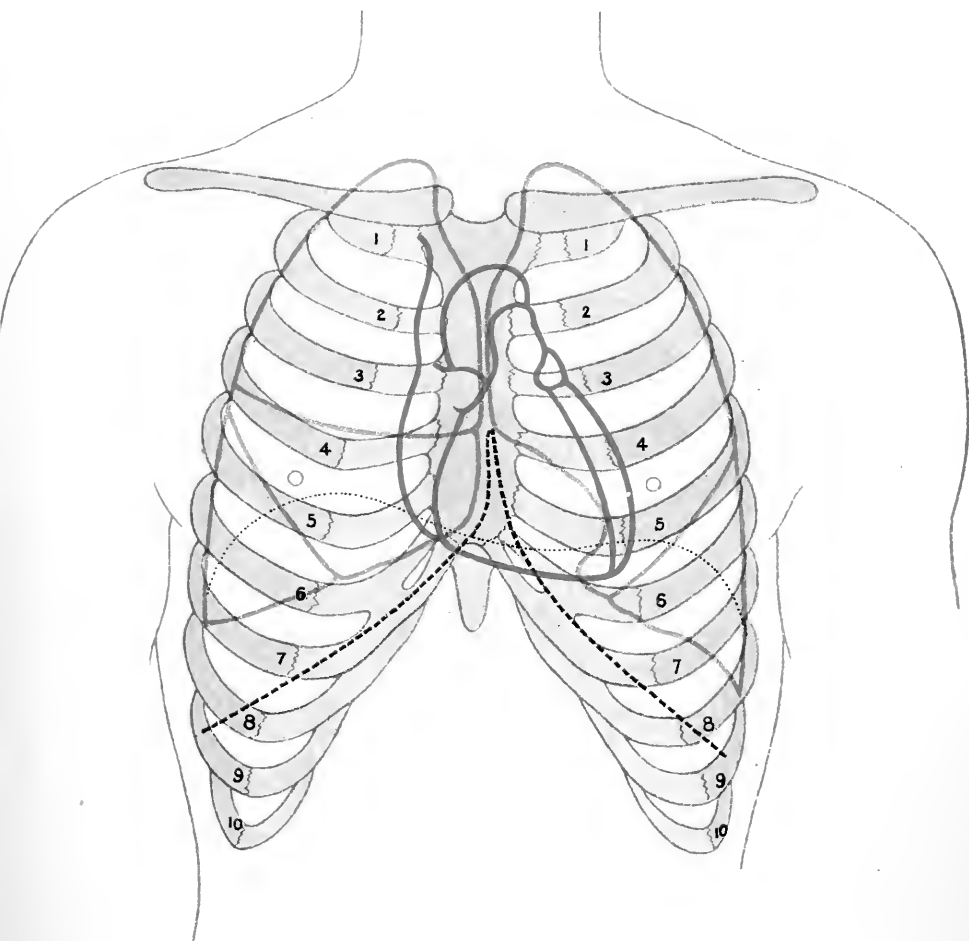


PLATE I (Front)

DIAGRAM SHOWING THE RELATIONS OF THE THORACIC VISCERA, ETC., TO THE SKELETON (kindly furnished to the Editor by Professor Thane).

The heart and great vessels are represented in red ; the outlines of the lungs and the great fissures are indicated by blue lines ; the lower limit of the pleura by heavy dotted lines ; the diaphragm by a thin dotted line.

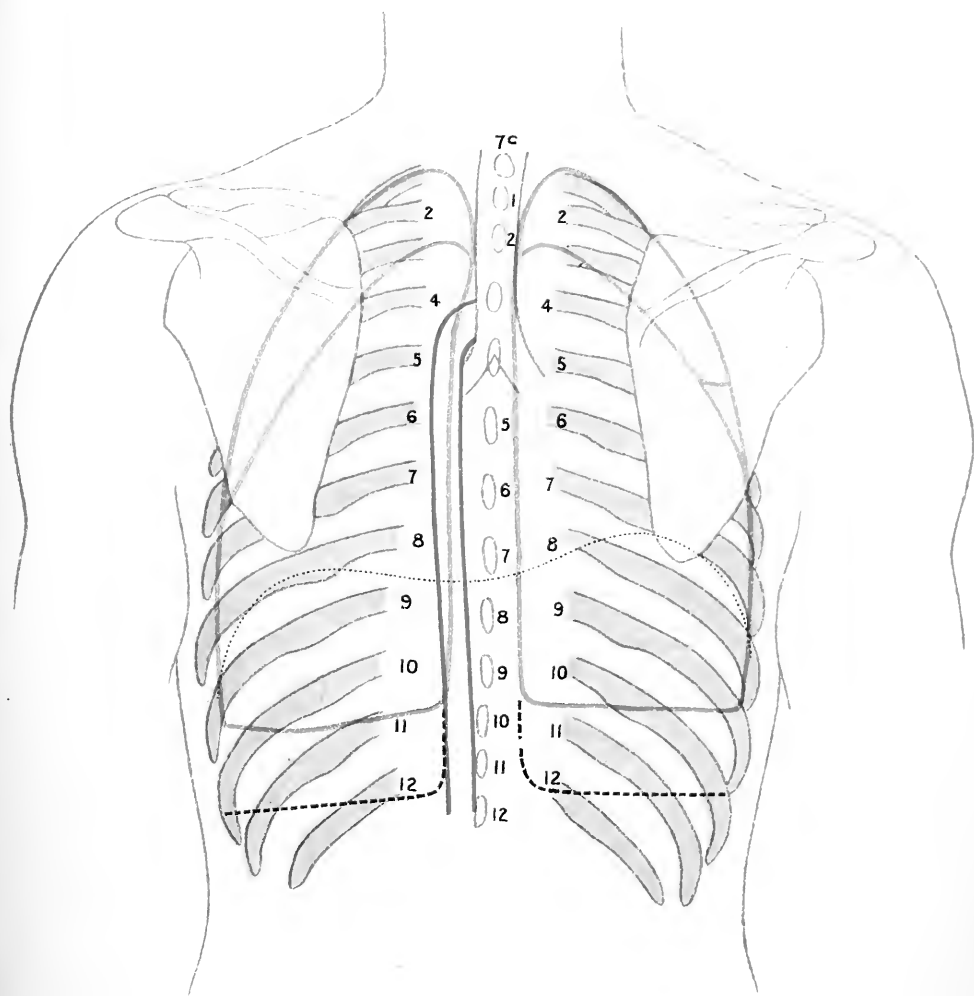


PLATE II (Back)

DIAGRAM SHOWING THE RELATIONS OF THE THORACIC VISCERA, ETC., TO THE SKELETON (kindly furnished to the Editor by Professor Thane).

The aorta is represented in red ; the outlines of the lungs and the great fissures are indicated by blue lines ; the lower limit of the pleura by heavy dotted lines ; the diaphragm by a thin dotted line.

Spines of VERTEBRÆ.	Bodies of	Original Spine	RELATION TO THE SPINES AND			
6C.	VI. C.	1				
	Disc.					
7	VII.	2				
	Disc.					
1D.	1 D.	3				
	Disc.					
	II.	4				
2	Disc.					
	III.	5				
	Disc.					
3	IV.	6				
	Disc.					
4	V.	7				
	Disc.					
5	VI.	8				
	Disc.					
6	VII.	9				
	Disc.					
7	VIII.	10				
	Disc.					
8	IX.	11				
	Disc.					
9	X.	12 D				
	Disc.					
10	XI.	2 an				
	Disc.					
11	XII.	4 an				

Cricoid cartilage

Commencement of trachea and gullet

ic duct (L.)

clavian artery

nnominate art. (R.) Inner end of clavicle

Upper edge of sternum

1st costal cart.

2nd costo-sternal artic.

Highest part of heart

3rd costo-sternal artic.

4th costo-sternal artic.

*Apex of heart 5th }
rest part of heart 6th } costo-sternal artic.
7th }*

Xiphi-sternal artic.

Adapted by the E.), the positions stated are either single in the median.
e size and shape of the chest, etc.

BODIES OF THE VERTEBRÆ

[illegible]

Adapted by the Editor from a similar table in Quain's *Anatomy* with the assistance of Prof. Thane; unless specified, right (R.), and left (L.), the positions stated are either single in the median line or bilateral; many of the positions are to be regarded as only approximate by reason of individual variations in the size and shape of the chest, etc.

THE RESPIRATORY SYSTEM—PHYSIOLOGICAL INTRODUCTION

The anatomy of the respiratory organs. The nasal air-way—the larynx—the chest—the air-tubes, pleura, and lungs. *The mechanism of respiration.* Inspiration—expiration—vital capacity—coughing and sneezing. *The respiratory exchange of gases.* The respiratory quotient—ventilation. *The blood gases.* The transit of the gases. *Cutaneous respiration.* *The nervous mechanism of respiration.* Eupnoea—apnoea—interrupted respiration—hyperpnoea and dyspnoea—asphyxia.

THE ANATOMY OF THE RESPIRATORY ORGANS

The nasal air-way.—The respiratory portion of the nose, *i.e.* the region below the middle turbinate bone, the accessory air-cavities in the nose, and the upper part of the pharynx, are lined with stratified ciliated epithelium. The tunica propria of the mucous membrane is formed of fibrillar connective tissue, infiltrated with lymphocytes. The lymphocytes wander out into the nasal cavity and here and there clump together in the mucous membrane to form solitary nodules. On the inferior turbinate bone the tunica propria is exceptionally thick (4 mm.) and contains many compound tubular glands. Deep and superficial capillary and lymphatic networks, and the terminal ramifications of the fifth nerve, lie within the tunica propria. On the posterior part of the inferior turbinate bone the veins are enlarged so as to form a cavernous tissue. The nose and upper part of the pharynx receive autonomic fibres from the seventh and ninth nerves (by way of the sphenopalatine ganglia), and from the cervical sympathetic. The former are probably vaso-dilator and secretory, the latter vaso-constrictor in function. By passing over the nasal mucous membrane, the air is rendered moist and warm, and is filtered free from dust and moulds. Inflammatory thickening of this membrane or other obstruction of the nasal passages leads either to over-action of the inspiratory muscles and consequent increase of the negative pressure in the thorax during inspiration, or to oral breathing

with exposure of the air-tubes to over-dessication, cooling, and irritation by dust. An infant is unable to suck when the nasal air is blocked. The openings of the Eustachian tubes and the posterior nares become obstructed by hyperplasia of the pharyngeal tonsils, and deafness results from the consequent absorption and rarefaction of the air in the tympanic cavity.

The larynx.—The true vocal cords, the anterior surface of the

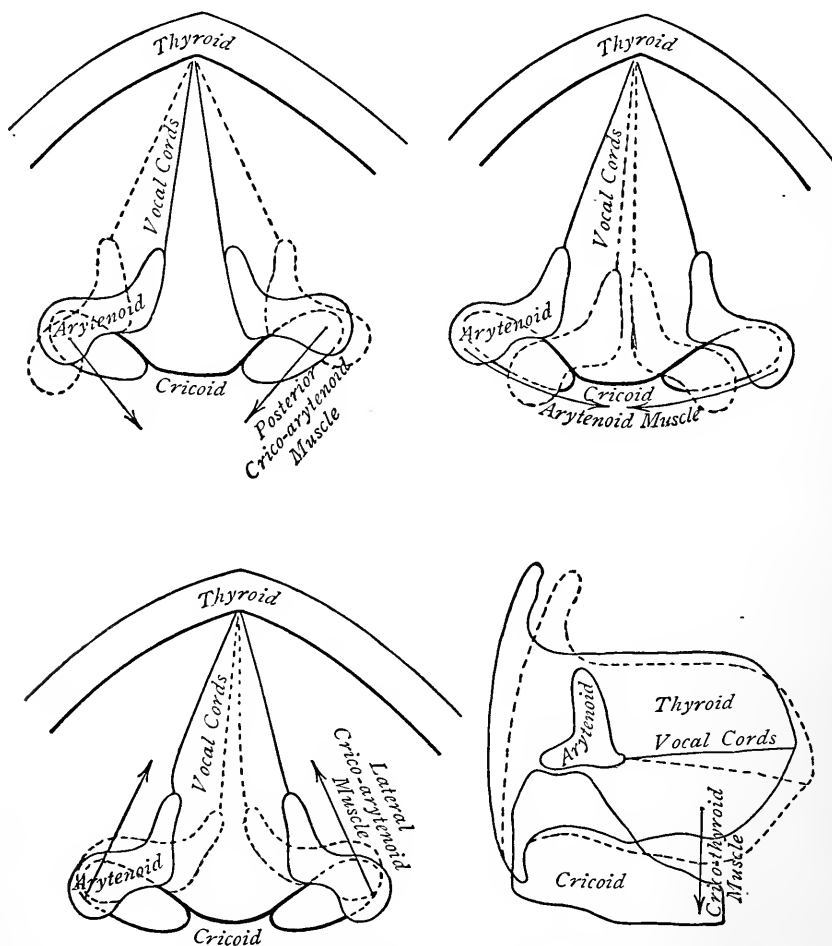


FIG. 1.—Diagrammatic representations of the actions of the laryngeal muscles. The dotted lines indicate the positions assumed by the cartilages and cords when acted on by the muscles contracting in the directions of the arrows. (After Landois and Stirling.)

arytenoid cartilages, and the laryngeal surface of the epiglottis are lined by a papillated and stratified scaly epithelium. The sinuses of Morgagni and the false vocal cords are clothed with ciliated columnar epithelium; the ciliary wave is directed towards the pharynx. In the submucosa there lie branched tubular mucous glands. Solitary nodules are also

found in the sinuses of Morgagni. The tunica propria contains numerous elastic fibres, and these form the greater part of the structure of the true vocal cords. The thyroid, the cricoid, and the greater portion of the arytenoids are composed of hyaline cartilage, while the epiglottis, the cartilages of Wrisberg and Santorini, the median portion of the thyroid, and the apices and vocal processes of the arytenoid cartilages are formed of yellow elastic cartilage. Between the twentieth and thirtieth year ossification begins in the thyroid and cricoid cartilages. The thyro-cricoid muscle, acting from the thyroid as a fixed point, rotates the cricoid, so that its posterior part, which carries the arytenoids, is depressed and further removed from the angle of the thyroid cartilage. By this means the vocal cords (thyro-arytenoid ligaments) are stretched. The lateral crico-arytenoid muscles rotate inwards the vocal processes of the arytenoid cartilages and adduct the cords; they are antagonised by the posterior crico-arytenoids. The arytenoid muscle is an adductor of the vocal cords, for it brings the arytenoid cartilages together. The thyro-arytenoid muscles are supposed to tighten or relax certain parts of the vocal cords and to alter the pitch of the voice. The thyro-cricoid is innervated by the superior laryngeal nerve, and the remaining muscles of the larynx by the inferior laryngeal nerve. The laryngeal nerve fibres arise from the lower bundles of the vagus. The larynx is controlled by a bulbar centre, and a higher adductor centre situated in the anterior part of the lower end of the ascending frontal convolution. The abductor muscles are normally maintained by the bulbar centre in a state of greater tone, so that the glottis during rest is slightly open. The cords tend to fall together and produce inspiratory obstruction when the inferior laryngeal nerves are divided. The adductor fibres are most excitable in the first stage of anæsthesia, while stimulation causes abduction of the cords when the anæsthesia is profound. The abductors are the first to fail through local action of ether, cold, etc. Autonomic fibres from the superior cervical sympathetic ganglia pass to the posterior part of the tongue, the pharynx, and the larynx, by way of the glosso-pharyngeal, the pharyngeal, and the superior laryngeal branches of the vagus. The superior laryngeal is the afferent nerve of the larynx. Stimulation of this nerve provokes expiratory spasm of the diaphragm (coughing).

The chest.—On the front the greater part of the thoracic wall is concealed by the pectorales majores. The upper parts of these muscles extend over the inner portions of the clavicles from which they arise. Inferiorly each muscle forms a prominent curved line which follows the direction of the fifth costal cartilage. The lower margin of the pectoralis major leaves the chest opposite the fifth rib and forms the rounded anterior axillary fold. The nipple is placed over the outer and lower part of the pectoral muscle, and generally lies between the fourth and fifth ribs and about four inches from the mid-sternal line. The upper

border of the pectoral muscle is at first separated from the anterior margin of the deltoid. There is thus produced the infra-clavicular fossa. The sternum is subcutaneous at the bottom of the sternal groove between the lines of attachment of the pectorales majores. A transverse ridge in the upper part marking the union of the manubrium and body of the sternum is partly produced by a forward projection of the second costal cartilages. The infra-sternal depression or epigastric fossa, a usually well-marked hollow, lies below the level of the seventh costal cartilages, and is placed over the ensiform cartilage. The upper margin of the sternum (in the expiratory position) is on a level with the disc between the second and third dorsal vertebræ. The junction of the manubrium and body of the sternum lies as a rule opposite the fifth vertebra and the xiphi-sternal articulation opposite the ninth vertebra. To the outer side of the pectoralis major the ribs are clothed by the serratus magnus. Below the pectoral muscle the thoracic wall is covered by the rectus abdominis and the external oblique. Posteriorly the latissimus dorsi ascends over the serratus magnus, and forms the thick posterior axillary fold. In a long narrow chest the lower ribs have an exaggerated downward slope and are very near to one another, the subcostal angle is narrow, and the lateral margin of the thorax reaches almost to the iliac crest. In a broad chest the opposite conditions are found. The costal angle formed by the seventh rib cartilages at the ensiform cartilage is normally about a right angle. All exercises in which the weight of the body is suspended from the arms tend to elevate and widen the chest.

The air-tubes, pleura, and lungs.—The apex of the *lung* ascends above the clavicle $\frac{1}{2}$ to 1 inch into the neck. It lies behind the interval between the two heads of the sterno-mastoid, and is covered by the subclavian artery and scalenus anticus muscle. The highest limit of the lung is on a level with the neck of the first rib. In inspiration the clavicle moves upwards and more or less covers the apex of the lung. From the apex the anterior border of the lung runs inwards to the junction of the manubrium with the body of the sternum, thence the two lungs descend together as far as the fourth costal cartilage. From this point the border of the right lung runs a straight or almost straight course to the level of the sixth sterno-costal articulation, while the left lung inclines outwards behind the fifth costal cartilage and leaves a triangular area of the heart exposed. This area is bounded by the mid-line of the sternum and by a curved line which runs from the sternal attachment of the fourth to that of the sixth left rib. The lower limit of the lung may be marked by a line carried round from the sixth sterno-costal articulation to the tenth dorsal spine. In the nipple line the lung extends to the sixth rib; in the line of the posterior fold of the axilla, to the eighth rib; in the scapular line, to the tenth rib. During the deepest inspiration the lung descends one rib lower down. The limits of the lung can be deter-

mined by percussion. The resonance of the percussion note is greater during deep inspiration, and is more evident in thin men. The area of superficial cardiac dullness is increased on expiration and diminished on inspiration. While on the left side almost the whole of the front of the chest is occupied by the superior lobe of the lung, on the right side the region above the fourth intercostal space is occupied by the superior lobe, the middle lobe occupying the lower part. The point of the inferior lobe just becomes visible on either side in the lower lateral part of the front view of the chest. Behind, the inferior lobes reach as high as the third dorsal spine, the right being usually rather lower than the left. The inferior lobes thus occupy almost the whole of the posterior view of the chest, with the exception of the supra-spinous fossæ, where the superior lobes become visible. "The septum between the left upper and lower lobes, starting from the third dorsal spine posteriorly, extends obliquely downwards and forwards, crossing the fourth and fifth interspaces, passing behind the scapula and sixth rib in the axilla to the upper border of that rib in the mammary line. On the right side the line of the septum terminates at the eighth rib, just outside the nipple line; and a second septum, starting behind the scapula, just external to the posterior fold of the axilla, runs transversely forwards along the fourth interspace to the middle line, thus forming the middle lobe."¹ The above statement is only approximately correct, for the lobes and septa vary somewhat in different subjects.

The lower posterior margin of the *pleura* always reaches the level of the eleventh dorsal spine, and may descend an inch lower than this point. The line of the pleura thence ascends to the sternum, passing behind the seventh costal cartilage, and reaching the mid-sternal line at the fifth sterno-costal articulation. The left pleura extends considerably farther over the pericardium than the corresponding lung. At the side of the chest the pleura usually reaches to within two or three inches of the lower margin of the thorax.

The *trachea* begins at the lower level of the cricoid cartilage. It is covered by the isthmus of the thyroid above, and below by the muscles and fat. At the upper margin of the sternum the trachea has receded from the surface to a depth of an inch and a half. The trachea is crossed by the arch of the aorta at the level of the fourth dorsal vertebra, and there bifurcates into the right and left bronchi. The posterior wall of the trachea is loose and flaccid, so as to allow of the forward expansion of the gullet. Stiffened by 16 to 20 hoops of cartilage in front and supported by the vertebral column behind, the trachea cannot be compressed except by the use of very considerable force.

The air-tubes are lined with ciliated columnar epithelium supported by a basement membrane. The meshes of the latter are continuous

¹ *The Localisation of the Lesions of Phthisis*, by Dr. J. Kingston Fowler, 1888.

with the lymph spaces of the mucous membrane. The ciliated cells are supposed to be regenerated from small oval cells which lie between their bases.

The tunica propria is composed of longitudinal networks of elastic fibres, and fine reticular tissue. The elastic tissue diminishes with advancing age. The hoops of cartilage in the trachea and bronchi are knit together by bands of smooth muscle, and the cartilages are embedded in connective tissue. Mucous glands and lymph nodules are plentiful in the submucosa, and are especially developed in the back of the trachea. A plexus of nerve fibres, containing small ganglion cells, and derived from the vagi and sympathetic nerves, supplies the muscle, the blood-vessels and glands of the air-tubes.

In the *subdivisions of the bronchi* the hoops of cartilage become broken up into segments, and these become smaller and finally disappear, as the bronchia subdivide into the bronchioles (diam. 0.5 – 1 mm.). Internal to the cartilages of the bronchia there is a well-developed circular band of muscle; this is innervated by the vagi nerves. In sections through the hardened lung, the mucosa of the bronchi appears thrown into folds by the contraction of this muscular coat. General spasm of the bronchial muscle greatly lessens the sectional area of the air-way, and leads to inspiratory dyspnoea, increase of the negative pressure in the thorax, and consequent over-strain of the elastic tissue of the lungs. The blood-vessels in the air-tubes form a deep stratum in the submucosa and a superficial capillary network beneath the ciliated epithelium. While the cilia cleanse the air-way, the cartilages maintain the patency of the tubes, the elastic tissue keeps their lumina free from wrinkles, and the muscular coat resists over-expansion during forced expiratory efforts.

In the smaller bronchi (diam. 1.0 mm.) the cartilage, muscle, and glandular tissue disappear, and in the bronchioles (diam. 0.5 mm.) the ciliated epithelium merges into one which is cubical, and contains patches of flattened scales. The elastic tissue continues over the surface of the alveoli.

The alveoli are produced by a pitting and infolding of the walls of the expanded ends of the bronchioles. The meshes of the capillary network are so closely set on the alveolar septa that the pulmonary epithelium appears to only fill up gaps in this network. Between the blood and the air in the alveoli there is interposed the scaly endothelium of the capillaries and that of the alveoli. The alveolar scales are non-nucleated, and both layers are of extraordinary tenuity. Groups of small granular and nucleated cells occupy the meshes of the capillary network. The walls of the alveoli are wet with lymph, which transudes through the inter-cellular cement substance. The endothelium is therefore permeated by gases in solution, and the permeability has been found to be greatly increased by distension of the lungs.

The terminal bronchioles, together with their arteries, veins, and

lymphatics, are knit together into lobules by reticular tissue and abundant elastic fibres. The interlobular connective tissue is continuous with the connective tissue coat of the trachea and air-tubes, and with the sub-pleural fibrous tissue, and this fact is of importance in connection with the extension of disease. The pulmonary veins are destitute of valves, while the arteries are exceedingly distensile and elastic. The pulmonary arterioles divide into capillaries which supply several contiguous air-cells; and thus if one arteriole be blocked by an embolus, the blood can reach the affected alveoli by the anastomosing capillaries. A venule arises from the base of each alveolus, and the contiguous venules anastomose with each other. The pulmonary tissues are supplied with arterial blood by the bronchial arteries, and the blood is returned from these by the bronchial veins which open into the right azygos, and left superior intercostal veins. The bronchial and pulmonary veins anastomose, and hence congestion of the pulmonary veins leads to bronchial congestion. The autonomic afferent nerves of the lungs enter the first, second, third, fourth, fifth thoracic nerves, and the lungs receive vaso-constrictor fibres from the same roots.

The total area of the alveolar surface is estimated to be 100 sq. metres, and the blood in the pulmonary capillaries may be reckoned to be exposed to the air over a surface equal to that of a room measuring 27 by 27 feet. The average time required for the blood to complete the pulmonary circuit is about 12 seconds. The average weight of the lungs is 1025 grms. About one-third of this weight is lung tissue, and two-thirds blood.

The lymphatics of the lung, sub-pleural, peri-vascular, and peri-bronchial, arise in the peri-alveolar lymph spaces, and, after traversing the small bronchial glands, find their way to the large glands at the root of the lung. Soot and dust particles, gathered by the lymph corpuscles from the air-passages, are lodged in the interlobular septa and in the lymph glands. The pleuræ are covered with scaly endothelium, and in this, especially opposite the intercostal spaces, are set numerous stomata. The pleural surfaces are moistened with lymph, secreted probably by the synovial fringes which project from the mediastinal wall of the pleural cavity. Watery solutions injected into the pleural cavity are rapidly absorbed by the blood-vessels.

The absorption of solutions from the air-tubes is also exceedingly rapid. Not only watery solutions, but cod-liver oil, creasote, and serum, are absorbed when injected into the trachea. The absorption is most rapid when the body is in the erect posture, and it is unaffected by section either of the pulmonary or phrenic nerves. The free inter-communication between the pleural cavity and the pulmonary lymphatics explains the extension of disease from the lung to the pleura. Pathological cases show that septic infection can travel from the throat down the loose external connective tissue coat of the trachea to the mediastinal

connective tissue and thence to the pleura and pericardium. It has also been demonstrated that infection can pass from the cervical lymphatic glands to the apices of the lungs—that is, in the reverse direction to the flow of lymph.

THE MECHANISM OF RESPIRATION

The thoracic cavity, when freely opened, appears to be far larger than its contents, for the lungs, owing to their elasticity, collapse so soon as the intra-pulmonary and pleural pressures become equal. In the fœtus the thoracic cavity is filled by its contents, but at the first breath of the new-born child the thorax assumes a new position; and as this is maintained by the increased tone of the inspiratory muscles, the lungs become permanently enlarged. As the thorax grows, and is pulled more and more into the inspiratory position, the elastic lungs expand to fill the greater cavity, and the intra-pleural pressure becomes sub-atmospheric by that amount of the atmospheric pressure which is required to overcome the elasticity of the lungs and distend these organs to the size of the thoracic cavity. The intra-thoracic cavity is a potential, not an actual space; and while the pulmonary and parietal pleural surfaces, with each respiratory movement, glide over one another, they remain in close apposition. The elastic traction exerted by the lungs on the thoracic wall, the heart, and thoracic blood-vessels—in other words, the intra-thoracic pressure—varies approximately as follows:—

Normal inspiration	— 10 mm. Hg.
Normal expiration	— 7 „ „
Deep inspiration	— 40 „ „
Deep expiration	0 „ „
Deep inspiration with air-way closed	— 100 „ „
Deep expiration with air-way closed	+ 100 „ „

The intra-tracheal pressure varies from -1 mm. Hg. in quiet inspiration to $+1$ mm. Hg. in expiration.

During forced expiration or inspiration with the glottis closed, the intra-tracheal is greater than the intra-thoracic pressure, by the amount of elastic traction exerted by the lungs. The pleural and pericardial sacs, the mediastinum, and all the structures enclosed therein, are affected by the respiratory oscillations of pressure. (The important influence of respiration on the circulation is discussed on p. 312.) When one pleural cavity is freely opened in a healthy man, the pressure becomes the same (atmospheric) within and without the lung. The stretched lung in consequence of its elasticity shrinks, and the mediastinum and organs therein are drawn over to the sound side owing to the elastic traction of the other lung. The total respiratory

capacity is thereby greatly reduced. When a valvular opening is made, the air is drawn into the pleural cavity at each inspiration, and fails to escape during expiration. Thus each breath increases the collapse of the lung, which finally becomes completely airless from absorption of the residual air.

The elasticity of the lungs is decreased by chronic disease, fever, and ill-nutrition, and diminishes with advancing age. Over-expansion, and consequent decrease of elasticity, results from either excessive dyspnoea, or forced expiration with the air-way obstructed, as in the lifting of heavy weights, coughing, or the blowing of wind instruments. Pleuritic adhesions, emphysematous destruction of lung tissue, pneumonic infiltration, lessen the distensibility of the lungs, while pleural and pericardial effusion, enlarged heart, increased intra-abdominal pressure, lessen their expansion. Tight-lacing especially impedes the action of the respiratory pump on the portal circulation.

If the elasticity of the lungs becomes unequally balanced on the two sides, the mediastinum will be drawn towards the lung which is exerting the greater elastic traction. In the case of pleural effusion the elasticity of the lung on the affected side becomes lessened by the collapse of the pulmonary tissue. Before the intra-thoracic pressure can become positive, it is necessary for the lung to be completely collapsed and its elastic traction exhausted. Thus in the first stage of pleuritic effusion the pressure of the fluid is never positive. The lung at first shrinks upwards from the diaphragm and then retracts towards the mediastinum, and when completely collapsed occupies the upper part of the hollow along the spine. So soon as this takes place, the fluid, if it still continues to be effused, begins to exert positive pressure and displaces the neighbouring organs.

INSPIRATION.—The distensile lungs follow at every point the inspiratory enlargement of the thoracic wall, and as the intra-alveolar pressure sinks below the atmospheric pressure, air and blood enter the lungs in increased volume. The vertical diameter of the thoracic cavity is increased by the contraction of the diaphragm. This dome-shaped muscular sheet flattens until the acute angle, between the thoracic wall and the diaphragm, becomes an obtuse angle. The lungs expand to fill this angle. The pull of the diaphragm on the lower ribs is antagonised by the pressure which the former exerts on the abdominal organs. At the same time the quadratus lumborum may fix the twelfth rib, and the serratus posticus inferior draw the four lowest ribs backwards. The central tendon of the diaphragm is slung to the pericardium and scarcely varies in position. In forced inspiration with obstructed air-way, or after a crushing lesion of the spinal cord below the origin of the phrenic nerves, the lower ribs are drawn inwards by the diaphragm and a girdle-like constriction is produced.

The antero-posterior and transverse diameters of the thorax are enlarged by the elevators of the ribs. The scalenus anticus and medius fix the first, the scalenus posticus the second rib. The external intercostal muscles, assisted by the levatores costarum and the inter-cartilaginous parts of the internal intercostals, taking the upper two ribs as a fixed point, elevate the thoracic cage. The levatores costarum, owing to the nearness of their costal insertions to the fulcra, produce a considerable movement of the sternal end of the ribs. By the elevation of the ribs the sternum is thrown forwards, the spine backwards, and the thorax is enlarged both from before back, and transversely. The effect of elevating each pair of ribs may be imitated by raising the handle of a pail from an angle of 45° to one of 90° with the pail. Each pair of ribs in the expiratory position inclines downwards from the spine to the sternum, and thus the sternal attachments are carried, during inspiration, not only upwards but forwards. The elasticity of the costal cartilages and the sterno-clavicular articulations permits this forward and upward movement. The angles formed by the ribs and their cartilages are at the same time straightened out, and this, together with the elevation and eversion of the ribs, increases both the transverse and antero-posterior character of the thorax. The movement of the upper ribs is chiefly forwards, and that of the lower ribs backwards, and this corresponds with the fact that the bulk of the lungs above lies in front, and below is behind.

If the pleural cavity be opened in the corpse, the thorax expands, for the elasticity of the lungs bends the thoracic cage inwards beyond the neutral point. It follows that in inspiration the muscles have only to overcome the elasticity of the lungs, for the thoracic cage will, so soon as this is overcome, spring outwards. In deep inspiration the upper ribs approximate, while the lower ones move further apart. The muscular energy expended in a deep inspiration is estimated to be equal to a pull of 500 lbs. As the thorax is very easily compressed in children, they die of cardiac syncope in the crushes of panic-stricken crowds. Artificial respiration is likewise more effectual in the young, for in them the circulation can be restored by rhythmical compression of the heart. In tight-laced women the upper part of the thorax gains an abnormal mobility. This thoracic type of breathing seems to be due largely to the wearing of corsets. Owing to its elasticity the thorax of the young adapts itself to local alterations of the elastic force of the lungs, and thus is pulled inwards in cases of phthisis, and bulges outwards in cases of pleuritic effusions or enlargement of the heart. By these changes the healthy lung is protected from over-expansion in phthisis, while in cases of pleuritic effusion the breathing space is not so seriously lessened.

EXPIRATION is brought about by the elastic recoil of the lungs, thorax, and abdominal wall, and occupies a slightly longer period than

inspiration. The transversales abdominalis with the oblique muscles, the recti and levatores ani acting as the natural "stays" of the abdomen, prevent both the dropping down of the viscera and the congestion of blood in the abdomen. In women the weakness of these muscles is a cause of dragging pain, weariness, and dyspepsia. The muscles can be developed by gymnastic exercises, and this is far more rational than the wearing of abdominal belts.

Extraordinary respiration.—In extreme dyspnœa every muscle is brought into play which elevates the ribs, or fixes the origin of muscles which elevate the ribs.

The patient grasps a support to fix the arms and shoulders, and while the serratus magnus, the pectorales, the latissimus dorsi, and the serratus posticus superior elevate the ribs, the serratus posticus inferior and quadratus lumborum fix the lower ribs, and antagonise the diaphragm.

In laboured expiration the abdominal muscles depress the thorax and at the same time force up the dome of the diaphragm. During deep inspiration the sternum becomes more convex forwards, the upper costal cartilages project beyond the sternum; and while at the back the angle of the ribs becomes more pronounced, in front the curve of these bones becomes straightened. These conditions are reversed in deep expiration. Owing to the bending of the thoracic cage, an elastic recoil takes place after both inspiration and expiration, and thus no time is lost, and the strenuous respiratory movements merge into one another without jolt or jar.

Breathlessness, resulting from muscular exercise, increases the capacity of the thorax. In ill-developed lungs many of the infundibula are collapsed, and these become opened out by deep inspirations. The inspiratory muscles are, moreover, excited to contract far more than the expiratory muscles, and thus the thorax, apart from the respiratory variations, is maintained in a tonic condition of enlargement during the whole period of exercise. As the sarcoplasm and tone of the inspiratory muscles increase with exercise, the thoracic cavity becomes permanently enlarged. During severe muscular activity the abdomen is drawn in by the tonic contraction of the abdominal muscles, and the diaphragm, its descent being resisted by the increased intra-abdominal pressure, raises the thorax. By this "pancostal" type of inspiration singers can execute a passage of $1\frac{1}{2}$ minutes, and sprint runners can run 100 yards on one breath. While over-strain imperils the elasticity of the lungs, too sedentary a life diminishes the size of the thorax. From the lack of physical development the clavicles, sternum, and scapulæ sink downwards, the ribs become very oblique, the costal angle acute, the diaphragm and heart elevated, the antero-posterior, vertical, and transverse diameters of the thorax diminished, the abdominal wall flaccid, and the viscera insufficiently supported. These conditions are reversed in athletes. Over-laborious work, such as the lifting of heavy weights,

tends to immobilise the thorax, for the cartilages of the latter become calcified, and the pulmonary elasticity decreases. The thorax is fixed more and more in the inspiratory position by the increasing tone of the inspiratory muscles. The onset of such a condition may be antagonised by expiratory exercises.

Measurement of thoracic mobility.—The thoracic girth in deep inspiration, and again in deep expiration, is usually measured at the level both of the nipples and xiphisternum. The average difference in girth is about 2 inches. The measurement is rendered inaccurate in muscular men by the contraction of the thoracic muscles. The lower is less than the upper measurement in adult civilised women owing to the wearing of corsets. The reverse is the case in men. The outline of the thorax may be obtained by modelling a flexible wire round the chest. The wire is divided behind and removed in two pieces, and these are fitted together on a sheet of paper and the outline traced.

The vital capacity is the greatest volume of air that can be expired after the deepest possible inspiration. This volume is measured by means of the spirometer. On the average it equals 3500 to 4000 c.c. (250 cub. in.). It is greater in men than in women, and in tall than in short men.

The *tidal air* is the volume of air breathed at each respiration, *i.e.* about 250 c.c. or 15 cub. inches. The spirometer is of value as an indicator of the effect of treating emphysema by expiratory exercises.

The intra-tracheal fall and rise of pressure, produced by the most forcible inspiration and expiration, can be measured by connecting a nasal tube with a spring or mercury manometer. The expiratory rise of pressure is especially diminished in emphysema, asthma, and bronchitis; while in phthisis, pleurisy, pneumonia, and in cases of laryngeal obstruction, the inspiratory fall of pressure is lessened. Fever reduces the variations of pressure by lessening both the muscular power and the pulmonary elasticity.

Respiratory sounds.—Accompanying the movements of respiration are certain sounds of peculiar character. On listening over the trachea or bronchi—most conveniently over the seventh cervical spine—there will be heard a sound lasting through the inspiratory act and divided by an appreciable break from another which continues throughout the expiratory act and is therefore rather longer than the former. Both sounds are of a quality *sui generis* which has to be learned, and are denominated “tracheal” or “bronchial.” They are of a harsh, blowing character, and are of much greater volume and of higher pitch than those to be next described. The expiratory sound is usually the more intense. These sounds are produced at the glottis, and are due to the vibrations into which the air is thrown by its passage through the glottis during inspiration and expiration (and probably in some degree by the entrance of the air through the nostrils and mouth), the vibra-

tions being communicated to the entire volume of air in the lungs and modified by resonance in the tracheal cavity.

The other normal respiratory sound, known as the "vesicular murmur," is audible wherever the lung is in contact with the chest-wall. It is heard throughout inspiration with a slightly increasing intensity, and continues on into the commencement of expiration without any separation or interval; but it does not last longer than the first third of expiration, and frequently may be scarcely audible at all during expiration. The vesicular sound is described as of a breezy quality, much less in volume than the bronchial sound, and of a soft, low pitch, the expiratory being fainter than the inspiratory. In children this normal vesicular sound is usually louder than in adults.

The cause of this vesicular murmur is not altogether clear, and several theories have been held. According to one view it is really the glottic sounds modified and damped down by transmission through

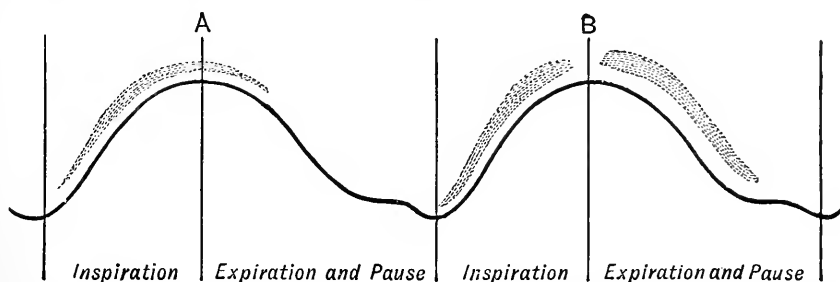


FIG. 2.—Tracings of the respiratory movements of a healthy male adult, reduced. The stippling indicates the sounds accompanying the movements of inspiration and of expiration. A, the vesicular sounds; B, the tracheal or bronchial sounds.

the countless subdivisions of the air-way formed by the bronchioles and alveoli. It is certain that these glottic sounds are ordinarily badly conducted to the surface of the chest, since among other things their intensity will vary inversely as the square of the distance from their seat of origin, viz. the larynx. Whether or not this be the explanation of the inspiratory element of the vesicular murmur, it is very probable that the conducted glottic sound constitutes the feeble expiratory portion of the same. The inspiratory portion has been explained otherwise. It has been shown by experiment and observation that sounds are produced by the passage of air through a tubular channel when the air moves with a sufficient degree of velocity through a narrower to a wider part of the lumen, eddies being set up and the movement of the air becoming turbulent, and not as a rule when it passes from a wider to a narrower part; though it may do so in the latter case when there is a projecting lip from the narrow orifice into the wide space. The opening of the bronchioles into the wider alveoli offers the condition suitable for the production of a sound; and inasmuch as it has been shown experimentally that suppression of the glottic sounds does not annul the inspiratory

sound audible in auscultation of the lungs, it seems probable that this part of the vesicular murmur results from the entrance of air into the alveoli themselves, and that this alveolar sound overpowers the glottic sound, and the more so the further the stethoscope is removed from a main bronchus. In the case of a rod or tube, such as the stethoscope, the law of inverse squares, such as pertains in the air, does not hold, and the sound is conducted almost without loss of energy. Another explanation has been given for the inspiratory sound, viz. that it is due to the separation of the moist surfaces of the alveoli by the inspirable air, comparable to the crinkling of a wet bag when distended.

Some writers describe, under the term "broncho-vesicular" breathing, as being a combination of the foregoing, the sounds to be heard over the roots of the lungs.

The larger vibrations of the vocal cords produced during vocalisation are propagated throughout the lungs and to the chest wall; when they become appreciable to the hand or ear of the observer as "vocal fremitus" or "vocal resonance." The former is much better marked in deep-voiced persons and less perceptible with high-pitched ones, as women and children. It is also rather more distinct in the right infra-clavicular region than in the left.

Coughing and sneezing mechanically keep the air-way open. In sneezing a deep inspiration is taken, and this is followed by a powerful expiration. The mouth is closed, the glottis in the first stage of expiration shut, and the air expelled explosively through the nose. The glottis is at first closed in coughing, and the intra-tracheal pressure raised by a forcible expiratory effort; the air is then suddenly expelled through the glottis and mouth. The soft palate, at this stage, closes the entrance to the nares, and the glottis is narrowed to a chink. The effect of coughing on the circulation is dealt with on p. 312. The diaphragm and abdominal muscles tend to drive the air from the lower to the upper lobes of the lungs, where the thoracic wall is protected by less powerful muscles. Chronic cough, therefore, diminishes the elasticity and distends the alveoli in the apices of the lungs.

The cough reflex depends on the integrity of the respiratory centre. It may be excited by cerebral conditions, but is usually aroused by irritation of the afferent fibres of the vagi. Irritation of the mucous membrane of the pharynx, larynx, and trachea, especially the posterior wall and bifurcation, excites coughing. Owing to the wide distribution of the vagi, coughing may likewise be excited from irritation of the pleura, stomach, liver, spleen, etc. Talking, singing, shouting, and crying increase the respiratory movements, expand the lungs, develop the muscles of respiration, prevent the congestion of blood in the venous system, and relieve the pent-up energy of the nervous system by the outward expression of the emotions. Yawning—a deep inspiration accompanied by tonic extensor spasm—expresses and drives into circulation

the blood and lymph, which stagnate in the tissues during a period of rest and immobility.

THE RESPIRATORY EXCHANGE OF GASES

	O ₂	N ₂	CO ₂
Average composition at 0° C. and 760 mm.			
Hg. of atmospheric air	20.96	79.02	0.03
Do. of expired air	16.03	79.59	4.38

Expired air is warmed nearly to body temperature, and from 50 to 100 Calories of heat are lost per diem in this way. It is saturated with water vapour (1 c.m. of air takes up 42.2 grms. of water at 37° C.). The total loss of water by the lungs varies from about 250 to 500 grms. per diem, and the heat lost by the evaporation of this water equals 145 to 290 Calories.

Of the tidal air (250 c.c.), one-third is breathed out from the large air-tubes at the following expiration, while two-thirds (about 170 c.c.) mixes by diffusion with the residual and reserve air (3500 c.c.) in the lungs. The composition of the alveolar air is thus altered by only one-twentieth of its volume at each respiration, and remains practically constant. The respiratory exchange of gases can be determined with considerable ease in man by Zuntz's apparatus. In an average man, weighing 70 kilos, the mean daily output of CO₂ equals 800 grms., and the mean intake of O₂ equals 700 grms. A half-metre cube roughly represents the volume of the intake of O₂ or output of CO₂.

Muscular activity greatly increases the respiratory exchange. In rest, walking (3 miles an hour), and hard labour on the treadmill the production of CO₂ is nearly proportionate to the numbers 2, 3, 6 respectively. A meal, a large draught of cold water, and a dose of sodium sulphate increase the respiratory exchanges by stimulating the muscular activity of the bowels. An indolent fat man may therefore be exercised by drinking purgative waters. Fatigued muscle works under unfavourable conditions, and is the seat of extravagant metabolism. A patient suffering from profound anæmia, when lying at rest, absorbs as much O₂ and excretes as much CO₂ as a normal man, also lying at rest. The anæmic patient, just as a balloonist in high altitudes, becomes breathless on exertion, for in him the reserve supply of O₂ is deficient. During a period of muscular activity the output of CO₂ generally exceeds the intake of O₂. In the subsequent period of rest the reverse is the case, and by the synthesis of oxygen with the muscle plasm a store of combustible material is formed. Man, being a warm-blooded animal, responds to a rise or fall in external temperature, with a decrease or increase respectively of metabolism and CO₂ output. The normal response occurs, however, only so long as the body temperature is normal. The

increase of respiratory exchange, which results from exposure to cold, is brought about by greater muscular activity, *e.g.* shivering, beating the chest with the hands, etc. A man deeply anæsthetised, or one in whom the spinal cord has been severed in the lower cervical region, behaves like a cold-blooded animal, and responds to a fall of external temperature by diminished metabolism and decreased bodily temperature. A new-born animal behaves like a cold-blooded animal for several days after birth. It is necessary, therefore, to protect the anæsthetised patient, the case of spinal paralysis, and the new-born infant from changes of external temperature. The CO_2 output is increased if the body temperature be raised by exposure to excessive heat or by fever. The temperature of Europeans in the tropics, being $.5^\circ$ to 1° F. higher than in the temperate zones, may increase metabolism and lead to a loss of fat.

The surface area of the skin, in proportion to the mass of the body, is much greater in the child than in the adult, and in the tall, thin man than in the short, fat man; consequently the loss of heat and the respiratory exchange are greater in the child and the tall, thin man.

The respiratory quotient $\frac{\text{CO}_2 \text{ output}}{\text{O}_2 \text{ intake}}$ indicates how much of the oxygen-intake is expended in the oxidation of carbon. While fat, *e.g.* $\text{C}_3\text{H}_5\text{O}_2$ ($\text{C}_{18}\text{H}_{35}\text{O}_2$), contains only one-eighth, and proteid less than one-half, of the oxygen required to combine with the hydrogen, carbohydrates, *e.g.* $\text{C}_6\text{H}_{12}\text{O}_6$, contain the exact proportion. Thus, as the proportion of fat and proteid are increased in a diet, the respiratory quotient becomes less. On an average mixed diet the respiratory quotient equals .8, while on a strict diabetic diet it is not more than .6. On the latter diet the intake of oxygen may be 50 per cent above that of the healthy man. Muscular activity increases the respiratory quotient, for the greater part of the energy is set free by the combustion of the carbohydrate moiety of the muscle plasm. In a starving man the energy is obtained from the proteids and fats of the body, and in him the respiratory quotient is decreased. The respiratory quotient may become greater than 1 when an animal is rapidly putting on fat. This is due to the conversion of carbohydrate into fat.

Ventilation.—There is no proof of the existence of any poisonous matter in expired air except CO_2 . The amount of CO_2 in even a very badly ventilated room is not sufficient to produce any symptoms. The excessive heat of crowded rooms interferes with the regulation of the body heat, and this is probably the chief cause of the nervous disturbance, aided by the smell which is due to exhalations from unclean bodies and clothes, walls and floors.

Rooms should be flooded with sunlight and freely ventilated, for not only are light, oxygen, and dryness destructive to many pathogenic bacteria, but, at the same time, light stimulates the play of nervous energy, while cool currents of air tone up the cutaneous blood-vessels,

increase the respiratory exchange and stimulate the deposition of fat. The ventilation of a room should be such that the CO_2 never rises above 2 parts in 10,000; and since expired air is warm and therefore rises, the inlets should be placed near the floor and the outlets in the ceiling. The ventilation of a closed room depends on the surface exposure of the walls, and this varies roughly as the square of the diameter of the apartment, while the volume of air in the room varies as the cube. A small room is thus better ventilated than a large one, and artificial means are required in the latter. In a closed room of 1000 feet capacity the air is completely changed at least once an hour, and the atmosphere is not vitiated by a single occupant by more than 0.5 per cent of CO_2 (Haldane).

Radiant heat should be used as a source of heat rather than hot air, for the latter is not only too dry, but, owing to its expansion, contains in a given volume less oxygen by weight. The frequency or depth of respiration has therefore to be increased in a room heated with hot air; and since the walls of such a room are colder than the air, the body may lose radiant heat.

THE BLOOD GASES

	O_2	CO_2	N_2	Measured at 0° C. and 760 mm. Hg. pressure.
100 vols. arterial blood				
yield	20-22	30-40	1-2	
100 vols. venous blood				
yield	10-14	45-50	1-2	

Oxygen.—Not more than 0.3 per cent of the oxygen is dissolved in the plasma, according to the law of partial pressures. Almost the whole of the oxygen is combined with hæmoglobin. Hæmoglobin forms 12.7 to 16.8 per cent by weight of the blood, and 1 grm. Hb. combines on the average with 1.25 c.c. O_2 at 0° C. and 760 mm. pressure.

The percentage oxygen capacity of the blood in man can be determined by the Haldane-Smith method. The oxygen capacity of 100 c.c. of sample ox-blood is first obtained by laking the blood and mixing it with potassium ferricyanide in a modified Dupré urea apparatus. The whole of the oxygen is thus displaced and measured. The blood of the patient (.05 c.c. is sufficient) is then compared colorimetrically with the ox-blood. The colouring power runs parallel with the oxygen absorbing power. In healthy men the percentage oxygen capacity is thus found to vary between 16 and 21. The total oxygen capacity and total mass of the blood in the body can be determined without much difficulty in patients by Haldane's CO_2 method (cf. *Journ. of Physiol.*, 1900). As a rule the mass of the blood in proportion to body weight is greater as the percentage oxygen capacity is less, so that the total oxygen capacity or total amount of hæmoglobin is relatively constant. In chlorosis the

volume of the blood is greatly increased, the percentage oxygen capacity greatly diminished, while the total oxygen capacity, deduced from these two factors, is about normal. In pernicious anæmia the total oxygen capacity is markedly lessened, in spite of an increased blood volume.

No free oxygen, or almost none, is to be found in the tissue lymph. If the atmospheric pressure be augmented, the free oxygen in the plasma increases by the law of partial pressures, until, when a pressure of five atmospheres is reached, the tissues become slowly poisoned, their metabolism and output of CO_2 diminished. Similarly the life of balloonists and mountaineers is endangered when the atmospheric pressure is lowered to less than one-half, although at this pressure the hæmoglobin is 97 per cent saturated with oxygen. It is clear, therefore, that the maintenance of a certain tension of oxygen in the plasma is a matter of supreme importance. The exchange of gases between the blood and the tissues cannot continue when the oxygen tension is five times greater or two-thirds less than normal. Loss of consciousness, without any premonitory symptoms of danger, results from exposure to an atmosphere containing too little oxygen. In foul wells the deoxygenated air comes out of the earth into the well when the barometer falls. Carbonic acid poisoning is practically unknown; an excess of CO_2 (over 5 per cent) produces dyspnœa.

Carbon dioxide.—About one-third of the CO_2 is carried by the red corpuscles and the remainder by the alkali of the plasma. CO_2 probably combines with the globulin moiety of the hæmoglobin molecule. In the tissues the tension of CO_2 equals 7 per cent of an atmosphere, and its mass influence is considerable, so that monophosphates and bicarbonates are formed in the plasma. In the lungs, as the tension of CO_2 falls to 4 per cent of an atmosphere, the mass influence of the phosphoric acid becomes greater, and diphosphates are formed, while the CO_2 is expelled. The proteids of the corpuscles, especially oxy-hæmoglobin, by their avidity for alkali, aid in the displacement of CO_2 . The ash of 1000 grms. of blood yielded in one experiment 4.34 grms. Na, of which 3.634 grms. were sufficient to saturate the chlorine, while the remaining .876 grms. combined as bicarbonate were sufficient to carry 632 c.c. CO_2 , or over 60 vols. per cent. In diabetic coma the alkalinity of the blood is neutralised by β -oxybutyric acid, and the CO_2 may fall to 3 vols. per cent. The amount of CO_2 in the secretions (bile, urine, etc.) varies with their alkalinity, and in pathological transudations is in inverse proportion to the amount of proteid. The latter acts as a weak acid. A large amount of nitrogen may occur in the bowels in certain cases of tympanites. This is due to air that is swallowed and to putrefactive processes. The nitrogen (unlike O_2 and CO_2) cannot be dissolved and carried away by the blood, for the latter is fully saturated with this gas at atmospheric pressure.

The transit of the gases between the alveolar air, blood, and tissues is usually represented as following the laws of diffusion:—

	Air.	Alveolar air.	Arterial blood.	Tissues.	Tensions per cent of an atmosphere.
Oxygen . . .	20.95	→ 18	→ 14	→ 0	
			Venous blood.		
Carbon dioxide .	.03	← 2.8	← 3.81	← 5.9	

The opinion of physiologists on this subject are, however, rapidly changing. Evidence has recently been brought forward which proves that the tension of CO_2 may be higher in the alveolar air than in the blood, and that the oxygen tension may be higher in the blood than in the air (Bohr and Haldane). In other words, the gases may pass through the pulmonary epithelium against tension. Oxygen is actually secreted in the swim-bladder of certain fishes, and this secretion is under the control of the vagus nerve. It is not only conceivable but probable that the passage of the gases through the lung may likewise be of the nature of a secretion. In the tissues diffusion may come into play, for the CO_2 tension is higher in the tissues than in the blood, while the O_2 tension is nil. Diffusion, however, is so slow a process that the exchange of gases cannot probably be attributed to it alone. The affinity of oxygen for hæmoglobin is maintained by the influence of the mass, *i.e.* the partial pressure of oxygen. In the tissues, where the partial pressure is nil, the oxy-hæmoglobin is reduced. The tissues combine with the oxygen so soon as it is set free.

That the chief seat of oxidation is in the tissues, and not in the blood, is evidenced by the fact that a frog, whose blood is replaced by salt solution, continues to move and produce CO_2 for many hours. The living tissues, moreover, deoxygenate methylene blue.

CUTANEOUS RESPIRATION.—The CO_2 excreted by the skin does not form more than $\frac{1}{100}$ th part of the whole, while the oxygen absorbed is still less. On obstructing the circulation in a limb, the cutaneous output of CO_2 is almost doubled, while that of water is slightly lessened. Dry dermatitis, produced by carbolic acid, diminishes the excretion of water by more than 50 per cent (Barratt). The whole skin can be covered with tar without producing any ill effect, for a man, unlike a fur-coated animal, continues to regulate his temperature under these conditions. The high mortality resulting from burns, which implicate more than a quarter of the total skin area, cannot be attributed to the arrest of the functions of the skin. The success which follows the treatment of patients suffering from burns with morphia and a single aseptic dressing suggests that shock of the nervous system is the primary cause of death in fatal cases. The concentration of the blood from loss of lymph is a secondary cause.

THE NERVOUS MECHANISM OF RESPIRATION

The respiratory centre consists of a bilateral tract, situated in the spinal bulb, in the neighbourhood of the nucleus ambiguus and deep origin of the vagus nerves. The centre can only be diffusely localised. Paralysis of respiration follows injury of the spinal bulb in this part. Afferent impulses are here switched over into efferent respiratory impulses; the two halves of the centre work synchronously together. The rhythmic discharge of respiratory impulses continues when the neural axis has been transected above and below the spinal bulb, as is evidenced by the rhythmic movements of the mouth and *alæ nasi*. The respiration becomes spasmodic and ineffectual when the neural axis is transected at the level of the pons and both vagi divided. The vagi regulate the normal respiratory rhythm. Each collapse of the lungs, by stimulating afferent fibres of the vagi in the lungs, reflexly evokes an inspiration, while each expansion of the lungs reflexly inhibits the inspiratory discharge. Expiration normally results from the elastic rebound of the thoracic and abdominal walls, and does not involve the activity of the neuro-muscular mechanism. If the vagi be divided, respiration continues at a slower rate, and at the same time becomes deepened. The vago-accessory bundle, arising from the nucleus ambiguus, contains the efferent fibres to the heart and bronchial muscles. The afferent respiratory fibres run in the lowermost of the upper bundles which form the origin of the vagus.

While weak excitation of the central end of the vagus nerve often provokes arrest of respiration in the expiratory phase, strong irritation causes inspiratory spasm of the diaphragm. Stimulation of the superior laryngeal nerve calls forth expiratory spasm, as in coughing. The respiration is reflexly inhibited during the act of swallowing by the glossopharyngeal nerve. Excitation of the central end of the splanchnic nerve provokes expiration, while a deep inspiration follows the application of cold water to the skin.

The rhythm of respiration is influenced by the slightest alteration in the cerebral circulation. Dyspnœa results equally from decrease of oxygen, from increase of carbon dioxide in the blood, from increase in the temperature of the blood. Asphyxial convulsions are produced by obstruction of the air-way, sudden loss of a large quantity of blood, or the production of acute anæmia of the bulb, *e.g.* by cerebral compression. The respiratory rhythm in the new-born animal continues for some minutes after removal of both heart and lungs, that is to say, after the abolition of the influence of the vagi and blood on the centre. It is probable, therefore, that the rhythm is, to a certain extent, an automatic function of the centre. The motor nerves are the phrenic and intercostal nerves, the inferior laryngeals supplying the larynx and the branches of the vagi which supply the bronchial muscles. The phrenic

nerves arise from the third, fourth, and fifth cervical spinal roots. They contain both afferent and efferent fibres. Paralysis of both phrenics renders respiration extremely difficult in man, and death soon ensues. A crushing lesion of the spinal cord above the origin of the phrenics is immediately fatal. The phrenic spinal nuclei are cross-connected. Death follows after both vagi have been divided in the young animal, from inspiratory obstruction of the air-way by the paralysed vocal cords; in the old, from septic pneumonia, due to the entrance of food-particles (through the anæsthetic glottis) into the air-tubes.

Eupnœa—the tranquil breathing of health. The frequency of respiration is 70-50 in the new-born child, 30-20 in the first five years of childhood, 20-15 in the adult. The ratio of respiration to pulse frequency is as 1 to 4-5. The inspiratory phase merges into the expiratory phase without any appreciable pause. The latter period is somewhat the longer. The frequency is increased by muscular activity, emotional excitement, sudden exposure to cold, elevation of body temperature; decreased during sleep. The respiration of the sleeper tends to fall into a grouped rhythm. This is especially the case with children.

Apnœa is the temporary inhibition of the respiratory act, which follows any voluntary attempt to breathe rapidly and deeply. The centre is inhibited, not so much by the over-oxygenation of the blood, as by the afferent impulses which pass up the vagi synchronously with each expansion of the lungs. Not only the reflex excitability of the centre, but that of the whole nervous system, is lowered during the condition of apnœa.

Interrupted respiration—groups of waxing and waning respiration—occurs naturally in sleep, and may become pronounced in conditions of deficient nutrition of the spinal bulb, *e.g.* in cases of cerebral anæmia, morphia poisoning, uræmia, etc. The group rhythm is probably due to the loss of the control which the higher centres of the brain exert over the respiratory centre. At the height of each group there often occurs a general spasmodic movement, accompanied by a partial return to consciousness. The pupil reflex and the blood pressure, as a rule, wax and wane synchronously with the respiration.

Hyperpnœa—excessive breathing—and **dyspnœa**—difficult breathing. Dyspnœa is excited by a cerebral blood supply deficient either in quantity or quality. Increase of CO_2 in the blood especially excites dyspnœa. In heart and lung disease it is the obstruction to the pulmonary circulation, as much as the want of air, that calls forth laboured respiration. By the action of the respiratory pump the blood is driven through the lungs.

Any sudden obstruction of the air-way reflexly excites the respiratory centre, for the pulmonary afferent nerves are excited by the abnormal diminution in intra-pulmonary tension which occurs during the ensuing inspirations. The respiration thus becomes deepened before the venosity

of the blood is increased. During obstructed breathing, the epigastric and supra-clavicular regions and the sides of the thorax are sucked inwards by the inspiratory diminution of intra-thoracic pressure. The period of inspiration is lengthened, for the expansion of the lung, which reflexly inhibits the inspiratory discharge, is brought about slowly. Similarly expiration is prolonged owing to the obstructed collapse of the lungs. Laboured respiration increases muscular work and CO_2 output; the partial obstruction of one bronchus leads to weak pulmonary breathing and slight thoracic expansion on the same side. A strident sound is produced at the seat of obstruction. On paralysis of the laryngeal abductors (posterior crico-arytenoids) inspiration becomes long, strident, and strenuous, while expiration remains free and short. Both the pulmonary circulation and the aeration of the blood continue when the volume of the lungs is very greatly diminished. The experimental compression of one lung does not alter the rhythm of respiration, while the output of CO_2 is maintained or even increased.

Asphyxia.—The condition resulting from complete obstruction of respiration may arise from (*a*) drowning; (*b*) exposure to an inert or irrespirable atmosphere; (*c*) obstruction of the air-way by hanging, impaction of a foreign body, tumours, diphtheritic membranes, etc.; (*d*) effusion of fluid into the lungs; (*e*) laryngeal spasm; (*f*) paralysis of the vocal cords in the young; (*g*) intoxication of the respiratory centre, as in morphia, chloral, chloroform poisoning; (*h*) paralysis of the respiratory centre by cessation of the blood flow; (*i*) coal-gas poisoning, whereby the oxygen is displaced from the hæmoglobin by carbon monoxide; (*j*) prolonged tetanic spasm, as in strychnine poisoning, tetanus, and status epilepticus.

After apnœa has been established by several rapid breaths, a man can hold his breath for 2 minutes, or, with distress, for 2 minutes 30 seconds: with practice this period may even be slightly extended. If the muscles be in activity this period will be greatly shortened. A man weighing 70 kilos. may be estimated to have 4000 grms. of blood. The arterial blood contains 18 per cent, and if in the apnœic condition all the blood were arterialised there would be 720 c.c. O_2 in the blood. Moreover, in the lungs, after a deep inspiration, there are about 4000 c.c. of air, and of this volume one-fifth or 800 c.c. may be taken to be oxygen. Air cannot be endured when the oxygen falls to 10-12 vols. per cent, so that only 320 c.c. of the oxygen in the lungs is at the man's disposal, together with 720 c.c. in the blood (total 1040 c.c.). The mean consumption of oxygen may be taken as 380 c.c. per kilo. per hour, or as 440 c.c. per minute in a man of 70 kilos. The man should therefore be able to hold his breath for a little over 2 minutes.

Strangulation very rapidly produces syncope, for the cerebral circulation is impeded by the occlusion of the carotid arteries. The reflexes persist for 2-3 minutes. Spontaneous recovery may occur after the

trachea has been obstructed for 3-4 minutes, but only $1\frac{1}{2}$ minutes after submersion of the body in water. The shorter time in the latter case is due to the inhalation of water. A person who passes into a syncopal state on immersion, may be recovered after a prolonged period, for in such case no water enters the lungs, and the heart, in the absence of struggling, is maintained for many minutes by the supply of oxygen in the blood. Recovery may be brought about by artificial respiration after the trachea has been obstructed for 6-8 minutes.

While convulsions hasten, anæsthesia prolongs the course of asphyxia. The temperature of the body exerts a marked influence. If the body temperature be lowered, the rate of metabolism is lessened, and the course of asphyxia prolonged. External cold, by hastening the rate of metabolic change, accelerates the onset of death in a normal heat-regulating animal. The new-born resist asphyxia for a long time. Hence the babe may be extracted alive from the womb after the death of the mother. The action of the vagi retards the onset of death in asphyxia by slowing and thus sparing the heart. After paralysis of the cardio-inhibitory mechanism by atropine, the tolerance to the asphyxial state is shortened by one-half. The body becomes gradually habituated to a process of slow asphyxiation, and owing to the concomitant fall of body temperature death takes place very gradually.

Symptoms of acute asphyxia.—*1st stage.*—The respirations become deeper and more ample. The heart is accelerated and beats with greater force. A feeling of agony supervenes. Muscular movements take place; these are involuntary, and almost convulsive in character. The ears hum and the world dances before the eyes. There follows a short stage of hallucinations, and then consciousness suddenly vanishes. The cortical cells are the highest and most unstable; they die the first, passing rapidly from intoxication to paralysis. The first stage lasts less than one minute.

2nd stage.—The reflexes are exaggerated. There are all the signs of agony, yet we know that consciousness is now in abeyance. The respiration is less frequent and of diminished force. At the end of the second minute the pupils dilate and there occurs emission of fæces and urine. The non-striped visceral muscles are thrown into contraction later than the skeletal muscles. The contractions are aroused by the anæmia of the central nervous system, but the latent period for non-striped muscle is long. The respirations at this period occur at rare intervals, the veins become congested with black blood, and the reflexes are abolished.

3rd stage.—The inspirations, which have occurred at longer and longer intervals, finally cease during the third minute. The heart now alone continues at work. The muscles and nerves are still excitable, but the bulbar centres controlling the heart and arteries are the only part of the central nerve system not yet paralysed.

The heart beats slowly, and with great force; the arterial pressure is high. If the vagi be cut during this period, the heart immediately accelerates. The heart's beats, remaining infrequent, gradually become reduced in strength; finally the heart accelerates and becomes more powerful. This is the last flicker before death, and recovery rarely takes place when artificial respiration is postponed to this moment. The final acceleration and the enlarged beat of the heart is probably due to the right auriculo-ventricular valves becoming incompetent, owing to the distension of the right ventricle. The acceleration also denotes the paralysis of the cardio-inhibitory mechanism.

The constriction of the visceral arteries that takes place in asphyxia drives the blood into the limbs and cutaneous vessels. The determination of the blood to the skin is perhaps for the sake of promoting cutaneous respiration, which is efficient in the amphibia. The salivary glands secrete during asphyxia and a cold sweat breaks upon the body. As the oxygen in the blood diminishes the sugar increases, and appears in the urine. Death arises purely from want of oxygen. The carbon dioxide in the blood does not rise to toxic amounts, and death occurs just as rapidly when respiration takes place in an atmosphere of nitrogen and the CO_2 is expired. The toxicity of the blood of one species of animal to another is said to be increased by asphyxia.

If the oxygen in the atmosphere be decreased below 20 per cent there occurs malaise; below 12 per cent, slow asphyxia; below 6 per cent, loss of consciousness and death. Dogs placed in an atmosphere containing 8-9 per cent O_2 sink into a condition of semi-coma. After four to five hours they pass bloody urine, and the red corpuscles of the blood are profoundly altered. If the O_2 be reduced to 5 per cent there occurs complete anuria, and the renal tubules become filled with hæmoglobin.

Recovery from asphyxia—Artificial respiration is the one and only method of recovery worth employing. The thorax should be rhythmically compressed over the region of the heart. By this means the blood is driven from the lungs into the left heart and thence on into the aorta. At the same time the lungs are ventilated. To drive on the blood and get arterialed blood into the coronary arteries and spinal bulb is the great object in view. Synchronously with the respiratory compressions, the tongue should be jerked forwards. This opens the larynx and, according to Laborde, reflexly excites the bulbar centres.

Complete recovery of the normal respiratory and cardiac rhythm and vaso-motor tone does not take place for one or two hours after asphyxia. The patient after resuscitation should therefore be kept quiet and in the horizontal position for some considerable time.

LEONARD HILL.

DISEASES OF THE RESPIRATORY SYSTEM

DISORDERS OF THE UPPER RESPIRATORY TRACT

Diseases of the nose have of late years been studied with such exactness and scientific accuracy as to justify their inclusion in a special branch of medical science—rhinology. Many of these affections, *e.g.* tumours and suppuration of the nose and its accessory cavities, are conventionally surgical in treatment, and hence a consideration of such finds no place in a text-book of clinical medicine. Nevertheless, there are some affections of the nose and naso-pharynx whose very frequency in every-day practice demands some mention in a treatise intended primarily for students and practitioners. It has already been mentioned that the nose, as the uppermost part of the respiratory tract, has important functions in the due performance of the respiratory act. Partial or complete arrest of these functions, as from pathological states of the nose, entails a risk of disease in other areas of the respiratory tract. The relations of the nose not only with the lungs, but with the pharynx, larynx, tympanic and cranial cavities, and the various accessory sinuses, indicate the possible widespread effects of nasal disease, and the importance of its early recognition and adequate treatment. In the following pages no attempt is made at classification or exhaustive description. A few of the commoner affections of the nose and naso-pharynx are alone dealt with, mainly from a clinical standpoint.

ACUTE NASAL CATARRH

SYN.: ACUTE CORYZA, ACUTE RHINITIS

Acute catarrhal inflammation of the nose and upper air-passages, popularly known as a “cold in the head,” is unpleasantly familiar to most people. It is rare to find an adult who has not experienced this common and annoying complaint.

Etiology and Pathology.—The actual causation of “catarrh” and the processes underlying its pathology are still imperfectly understood. The precise mode of origin of a “cold in the head” still shares the mystery which shrouds the birth of a “bilious attack.” Exposure to sudden changes of temperature, whether

from hot to cold, or *vice versa*, is undoubtedly the most constant antecedent factor. The various methods in which this "chilling" of the whole or a part only of the body surface obtains (including draughts, wet feet, etc.) are too familiar to call for reiteration. One of the most striking features in the etiology of coryza is found in the very marked *rôle* which individual susceptibility plays. One person may expose himself without after consequences to draughts or repeated wettings, while another in apparently equal health "catches cold" on the slightest provocation. This idiosyncrasy must obviously depend on some inherent property of tissue, which for want of a better term is designated "diminished powers of resistance," and such patients are often said to be "delicate." Pathological conditions of the nasal cavities, such as chronic inflammations or the presence of "spurs," over-exhaustion either of body or mind, and heredity, are all cited as predisposing causes tending to precipitate an attack. It has been claimed that all these act by exerting a "lowering" effect, which lessens the resistance of the nasal mucous membrane to some specific micro-organism. But although various well-known bacteria have been described as existing in the nasal secretion in acute coryza, no specific *materies morbi* has yet been recognised. The sudden onset, course, and undoubted contagiousness of a common cold would certainly suggest a specific cause. It is to be noted also that a "cold" or "chill" may affect other mucous membranes besides those of the respiratory tract. Gastric symptoms, such as epigastric pain with severe vomiting, intestinal trouble evidenced by abdominal uneasiness and irregular violent diarrhœa, are examples of results which may follow a "chill."

It would seem that in individuals so predisposed, now one and now another organ may be the seat of the main stress of the catarrhal process. Further discussion of these considerations would be out of place in the present article; enough has been said to indicate the ill-understood nature and the widespread effects of a "common cold."

Acute nasal catarrh is commonest in young persons. The inhalation of irritant vapours may bring about a similar condition, which disappears on removal of the cause. It must be remembered that in children especially identical symptoms may herald the onset of measles, and less commonly influenza, or may follow the administration of the iodides of potassium or sodium. Glanders, the "snuffles" of infants suffering from congenital syphilis, and nasal diphtheria are instances of nasal trouble which only the most superficial observer could confound with acute coryza.

Symptoms.—These are too well known to demand more than a brief outline. The onset is usually sudden, with chilly feelings or repeated sneezing, there is frontal headache with slight pyrexia, and the temperature may rise to 100° or 101° . The head feels “full,” and the nose is hot, dry, and “stuffed-up,” the back and limbs may ache, there are all the usual accompaniments of slight fever, and the patient feels indisposed rather than acutely ill. The nasal discharge at first is thin and scanty, but in a few hours becomes copious and acrid. The pocket-handkerchief is in annoyingly frequent demand, and the nasal apertures and adjacent upper lip tend to become sore, red, and inflamed. Herpetic vesicles about the lips are sometimes seen, and often recur in the same individual in subsequent attacks. The swelling of the nasal mucous membrane at this stage is usually sufficient to cause nasal obstruction, and the necessity for mouth-breathing adds to the general discomfort. The sense of smell is blunted or temporarily lost, and hence also there is no appreciation of flavour. Extension of the inflammatory process explains such frequent symptoms as throbbing frontal headache from implication of the frontal sinuses, and epiphora from swelling of the naso-lachrymal canal. In a similar way pharyngitis with painful swallowing, or subacute laryngitis with hoarseness, may occur, and a temporary deafness or tinnitus often results from involvement of the mucous membrane of the Eustachian tubes. The voice is altered, and such consonants as *m*, *n*, *ng* are malpronounced; “man” becomes “bad,” “common” sounds “cobbod.”

As a rule, the acute stage begins to subside within thirty-six hours, the nasal discharge becomes muco-purulent and gradually diminishes, and the symptoms disappear within from three to six days. If ordinary common-sense precautions are taken, it is rare to see any serious after-effects from a common cold. Occasionally the affection tends to become chronic, or as the nasal symptoms subside, the larynx, trachea, and bronchi may become successively affected, and a more or less troublesome bronchial catarrh results.

Acute rhinitis sometimes occurs in young infants, and it should be remembered that such cases may be gonorrhoeal in nature. In addition, the nasal passages in an infant are so narrow that a comparatively slight rhinitis may cause complete nasal obstruction with serious impediment to respiration and the act of sucking.

Morbid Anatomy.—The mucous membrane of the nasal cavities shows swelling and hyperæmia with increased secretion. A similar condition exists in the other mucous membranes to which the process may spread. The nasal secretion is usually slightly

alkaline, contains traces of albumen, yields with acetic acid a precipitate of mucin, does not reduce Fehling's solution, and has a peculiar "stiffening" effect when drying upon the handkerchief.

Treatment.—This is best discussed as (1) Preventive, (2) Abortive, (3) Palliative.

Preventive Treatment.—The personal equation is a striking feature in most problems of disease, and the varying liability of individuals to acute nasal catarrh has already been pointed out. One patient goes through life "always catching cold," while another under almost identical surroundings endures exposure or cold with successful equanimity. Heredity, the presence of any chronic nasal trouble, and a sedentary life, are all credited with a share in the curious proclivity to "cold-catching" undoubtedly possessed by some individuals. Apart from idiosyncrasy, there is no doubt but that much can be done to avoid acute nasal catarrh. Draughts, sudden changes of temperature, and such follies as sitting in wet garments must of course be avoided. Suitable clothing is most important, and it should be noted that to be habitually overclad is as noxious as the opposite extreme. Underclothing should be of wool or silk, and "cellular" materials are highly recommended by some authorities. All forms of "coddling" must be discouraged, and such customs as the wearing of handkerchiefs round the neck probably do more harm than good. Adequate ventilation must be insisted on, and the common custom of sleeping with closed windows abandoned. The regular morning "tub," either cold or just tepid, followed by a brisk "rub-down," is one of the best preventives of habitual cold-catching. Bare arms and legs in weakly children in cold seasons should be covered. In adult men the cultivation of a beard in place of shaving sometimes acts like a charm.

Abortive Treatment.—Can an acute coryza be aborted or cut short? is a question often asked, and it must be confessed not easily answered. It would be difficult to find a household in which some remedy is not held in almost religious respect as a specific in the arrest of a "common cold in the head." Morphine, atropine, a Turkish bath, the inhalation of eucalyptus oil, repeated doses of camphor, quinine, or belladonna, and the application to the interior of the nose of menthol, cocaine, Ferrier's snuff (containing bismuth and morphine), or the various carbolated or other "anticatarrh" smelling-salts, are a few of these better-known "cures." It may safely be said that no one method of treatment can always be relied upon, and not a few of the remedies advised are themselves worse

than the disease. Immediately symptoms develop, a hot bath and 10 grains of Dover's powder given in hot whisky-and-water directly after getting into bed offer the best chance of bringing a common cold to a speedy conclusion.

Palliative Treatment.—When a severe coryza is definitely established, it is wise for the patient to remain indoors in a warm room. Ordinary diet may be taken, and the restriction of, or even abstention from, liquids recommended by some authorities only adds discomfort to the already overburdened individual. Tincture of aconite in one-minim doses every hour may relieve the throbbing headache, and the inhalation of steam or the fumes of ammonium chloride through a suitable inhaler sometimes alleviates the “nose-stuffiness” which all patients complain of. In some cases menthol undoubtedly gives relief, and is conveniently used in the form of Cushman's menthol inhaler. The local application of cocaine in 4 per cent solution has been strongly recommended, but the very real danger of establishing the cocaine habit through its use in a comparatively trivial affection must never be lost sight of. In the large majority of cases no drug treatment is called for, and the patient may even continue his ordinary avocation. In the weakly or debilitated convalescence may be slow, and tonics such as Easton's syrup indicated, while a change to some bracing seaside resort is frequently the surest means of restoring the patient to robust health.

EPISTAXIS

Epistaxis or nose-bleeding is an exceedingly common symptom, and but few individuals reach maturity without some experience of a complaint more often annoying than serious. Of all the mucous surfaces, the nasal mucous membrane is most liable to spontaneous, or apparently spontaneous, hæmorrhage. This is not surprising when the structure of the nasal membrane and the wealth and arrangement of its blood-vessels are remembered. These vessels are very numerous and ill-supported. Over the middle and inferior turbinate bones the veins form a rich venous plexus, and the mucous membrane “assumes the character of cavernous tissue, and is sometimes spoken of as the ‘erectile body’” (Cunningham).

During the eight years 1893-1900 (inclusive) no less than 149 patients were admitted into the London Hospital suffering from severe epistaxis which ordinary methods had failed to arrest. Epistaxis is comparatively rare in infants, but is commonest between

the ages of 2 and 16. It is less often seen in adults, but becomes again increasingly frequent as age advances and degenerative changes take place in the blood-vessels. Males are said to be more often affected than females; and of the 149 cases mentioned above, 89 were males and 60 females.

Etiology.—Epistaxis may result from very widespread causes. These are best considered under three groups. *A. Traumatic; B. Local; C. General.*

A. Traumatic causes. The most familiar example under this heading is the hæmorrhage, usually temporary and smart, which follows a violent blow on the nose. Fracture of the nasal bones is usually accompanied by some hæmorrhage. In fractures involving the anterior fossa of the base of the skull, bleeding from one or both nostrils is frequently seen. Slighter causes, such as violent use of the handkerchief, the passage of instruments, or nose-picking in children, may set up slight epistaxis.

B. Local causes.

1. Local inflammation.—Occasionally in acute or chronic rhinitis there may be a little nose-bleeding.

2. Local ulcerations.—All ulcerations of the nasal mucous membrane, whether tuberculous, syphilitic, lupoid, diphtheritic, or set up by a foreign body, may give rise to epistaxis.

3. New growths.—Benign growths, as polypi or papillomata, very rarely bleed, but hæmorrhage from malignant new formations is common.

C. General causes.—These form a very important and comprehensive list. From a clinical standpoint they may be considered under the following groups:—

1. The onset of acute specific fevers.—Repeated slight epistaxis is now well recognised as a frequent early symptom in typhoid fever. Less often it occurs in the early stages of measles, variola, influenza, and relapsing fever; also in the course of rheumatism.

2. Blood diseases.—In conditions in which the blood-constitution is profoundly altered, hæmorrhage from the nose is common. Severe simple anæmia, pernicious anæmia, splenic or lymphatic leukæmia, purpura, scurvy, and hæmophilia are instances in which repeated nose-bleeding may occur at any stage of the disease.

3. Conditions of high arterial tension.—The best example of this group is seen in the repeated epistaxis so often occurring in the later stages of cases of granular kidney. Thus repeated nose-bleeding is common as an early symptom of uræmia.

4. Degenerated blood-vessels.—In arteriosclerosis, atheroma, or similar degenerative disease of the arteries, epistaxis not rarely occurs on very slight provocation.

5. Obstruction in venous return from the head.—In such conditions as chronic valvular disease of the heart (notably mitral stenosis and aortic regurgitation) or chronic pulmonary affections (*e.g.* old standing emphysema with bronchitis), repeated nose-bleeding is common. During the paroxysms of whooping-cough it is equally prone to occur. Direct pressure on the large venous trunks of the neck, as by a bronchocele or other neck tumour, has sometimes set up severe epistaxis.

6. Idiopathic.—Under this somewhat meaningless heading must be separately mentioned a large and common set of cases in which repeated attacks occur without discoverable cause. Such patients are usually young adolescents, and in not a few a definite hereditary predisposition would seem to exist. The hæmorrhage is rarely serious, and as a rule ceases spontaneously.

7. Miscellaneous Conditions. — These include certain drugs (phosphorus, the salicyl compounds, etc.), rarefied air as in mountaineering, and chronic liver affections, notably cirrhosis. Some authorities still believe that epistaxis may replace the menstrual flow, and the literature contains various well-recorded, if not convincing, cases. It is always difficult to exclude the possibility of coincidence in these, and vicarious menstruation must still be regarded as “non-proven.”

Finally, it must be remembered that in many of the above conditions the immediately exciting cause is often some strain, such as the use of the handkerchief or violent sneezing, which in a healthy subject would bring about no such untoward result.

Symptoms.—Whatever its cause, the blood may come from both nostrils, more often from but one. Its origin may be either arterial or venous. The mode of onset varies: it may be sudden and continuous, or repeated and slight. As a rule it persistently “drips,” sometimes it “runs” in a more or less steady stream, but “spiriting” of arterial type is very rare. The quantity varies from a few drops to several pounds. The colour is usually bright-red, and it clots with great rapidity: curious pendent tumours of clotted blood are sometimes thus formed. The varied conditions mentioned above as “general” causes obviously act by predisposing the vessels to bleed on very slight provocation. In a surprisingly large number of cases the actual bleeding site is found to be “a spot about half an inch from the anterior end of the cartilaginous

septum" (Dr. de Havilland Hall). This has been called the "site of predilection."

Diagnosis.—Under this heading two points only call for notice.

1. *The blood may come from elsewhere and be transmitted only through the nose.*

A fracture of the anterior fossa of the skull with injury to some vessel, a similar accident to or growth of any of the various accessory cavities communicating with the nose (*e.g.* nasopharynx), may any of them lead to severe hæmorrhage from the nose.

Such condition must be distinguished from true epistaxis.

2. *True epistaxis may lead to discharge of blood from other channels.*

(a) The blood may enter the pharynx and be swallowed, to be vomited later with the characteristic "coffee-ground" appearance of partially digested blood. This is especially common if epistaxis start during sleep. "Coffee-ground" vomiting on first waking should always lead to a suspicion of nose-bleeding as the cause.

(b) The blood may enter the larynx, excite cough, and be discharged with sputum. This is always difficult to distinguish from hæmoptysis. In such cases a careful examination of the nose and nasopharynx will often reveal the cause of what may otherwise be attributed to grave pulmonary disease.

Prognosis.—This will obviously depend upon the cause. In a large number of cases of epistaxis the bleeding is comparatively trivial, and either ceases spontaneously or is arrested by simple measures. It must be remembered that the blood lost may be sufficient to cause severe anæmia, and fatal cases in which the hæmorrhage has persisted in spite of all treatment are by no means unknown.

Treatment.—Sir Thomas Watson long ago pithily wrote that epistaxis is "sometimes a remedy, sometimes a warning, and sometimes a disease in itself." Hence active treatment directed to its arrest may be not only unnecessary but positively harmful. This is notably the case in such diseases as granular kidney with high blood tension, when a smart epistaxis may relieve the circulation to such an extent as to replace or ward off such disasters as uræmic convulsions or cerebral hæmorrhage. On the other hand, many cases cease spontaneously and are so slight as to require no active treatment. In every case of nose-bleeding it should be a cardinal rule to make careful examination as to the probable cause before entering upon any treatment. Constitutional remedies suitable to the cause will be called for in many cases, but such cannot be

discussed here. When it is thought wise to attempt the arrest of the hæmorrhage, there is the choice of a large number of remedies. Such simple measures may first be tried as rest, elevation of the arms, application of ice to the bridge of the nose, cold to the upper part of the spine, or placing the feet and legs in water as hot as can be borne. These often alone suffice. In other cases, especially when the bleeding comes from what has been called the "site of predilection," pressure applied by the patient's finger to the corresponding ala nasi against the septum is effectual. Thorough syringing of the nasal cavities either with ice-cold or hot water may be worth a trial. The application of astringents such as cocaine, hammamelis, or perchloride of iron may sometimes be of service, but when applied indiscriminately to the whole of the interior of the nose they frequently do more harm than good. Should simpler measures fail and the bleeding point have been detected, the application to it of the galvano-cautery is a very efficacious mode of treatment. A blunt "point" should be used at a dull-red heat. If the hæmorrhage continue, the nose should be plugged. Either the anterior or posterior nares or both may be thus treated. Plugging the anterior nares is simple and easy. A single strip of iodoform gauze packed in through a speculum is the best method. Posterior plugging is more difficult and is apt to be unpleasant to patient and practitioner alike. Bellocq's sound or a soft rubber catheter is necessary; but for a detailed description of the *modus operandi* the reader is referred to one of the many surgical manuals.

It should be noted that this measure is seldom really necessary, and that when adopted the plug should not be left *in situ* for more than twenty-four hours, since septic troubles are very prone to follow its retention. Recently the local application of a strong solution of suprarenal extract has been highly praised as a means of arrest of uncontrollable oozing from the nasal mucous membrane.

ADENOIDS

SYN. : ADENOID VEGETATIONS, POST-NASAL GROWTHS, HYPERTROPHY OF THE PHARYNGEAL TONSIL

The existence of lymphoid or adenoid tissue in the nasopharynx has been known since the days of William Hunter, but it is only in comparatively recent years that its due importance and extent

have been fully appreciated. During the period of active growth when infancy merges into childhood and childhood passes into adolescence, lymphoid tissue is widely distributed in various parts of the body. Its aggregation in the nasopharynx into a more or less defined mass has been picturesquely called the pharyngeal or Luschka's tonsil. The affection known as "adenoids" consists in a true hypertrophy of this lymphoid tissue found in the pharyngeal vault. Meyer of Copenhagen in 1868 first realised its importance, and gave a remarkably full and complete account of its pathology, symptoms, and treatment.

Etiology. — Amidst much that is doubtful, age is the most constant factor in the etiology of adenoids. All authorities agree that they are most common between the ages of five and fifteen, *i.e.* at the period of life when growth and development are most rapid. It would appear that at this time lymphoid tissue exists in greatest abundance, and we might therefore expect that slight causes may suffice to excite its overgrowth. Adenoids have been described in infants of but a few weeks old, and in some cases have been thought to be present at birth. They are rare, but by no means unknown, in adult life. Like lymphoid tissue in other parts, adenoids tend to shrink and atrophy after the age of 25; but the disappearance of symptoms after that age is doubtless helped by the relatively smaller space they occupy in the more widely developed pharyngeal vault. Sex would appear to have but little determining value, and they are probably as common in girls as in boys. A damp and cold climate was at one time held as an important predisposing cause, and adenoids were described as rare in warm countries. Later investigation tends to show that they may be found in all climates. They are particularly common among the Jews. It is not easy to estimate the influence of heredity: on the one hand, it is common to find several children affected in a family whose parents have had adenoids in their youth; on the other hand, the extreme frequency of the trouble and exposure to a common cause in the environment may serve to explain such cases.

The actual exciting cause can rarely be ascertained. Repeated catarrh from cold, unhealthy surroundings necessitating existence in an impure and de-oxygenated atmosphere, and such specific diseases as measles, scarlet or typhoid fever, are common antecedents. In this connection it is interesting to note the undoubted growth of adenoids which occurs in some cases of secondary syphilis and lymphatic leukæmia, diseases in which the lymphoid tissue

scattered over the body as lymphatic glands is markedly affected. A large proportion of children with cleft palate are found to have adenoids: the direct irritation of food and cold air which this deformity entails has been held responsible for such occurrence. Hypertrophy of the faucial tonsils commonly co-exists with adenoids. The faucal tonsils are mainly composed of lymphoid tissue, and any cause, or combination of causes, exciting hypertrophy of this tissue in the nasopharynx may reasonably be expected to bring about their enlargement also. Little is definitely known as to the exact physiological function of the lymphoid tissue so freely distributed in the upper part of the respiratory tract. Its aggregation in such considerable masses as the faucal and pharyngeal tonsils at the main entrance of inspired air would in itself suggest some important duties. It is probable that this lymphoid tissue furnishes leucocytes which serve to destroy or in some way neutralise the effects of the various micro-organisms so prone to enter with the inspired air. Disease of these organs would imply loss of this important function, and the enlargement of cervical glands commonly seen in cases of adenoids is evidence in the same direction.

Morbid Anatomy and Pathology.—Adenoids may be confined to the roof of the nasopharynx, but are usually found also to some extent on its lateral walls. The growth of lymphoid tissue is usually irregular, giving rise to smooth rounded masses, either sessile or pedunculated, varying in size from a hemp-seed to a currant, though larger projections up to the size of an almond are by no means rare. Less often the hypertrophy is more regular, and a flat raised “cushion” replaces the normal pharyngeal tonsil. The colour varies from pale pink to red, and seen *in situ* their appearance has been aptly compared to that of stalactites.

Under the microscope adenoids show the usual appearances of lymphoid tissue: a fine reticulum crowded with lymph corpuscles with a slight and varying proportion of connective tissue. This is arranged as a rule in a more or less lobular manner, and is covered on its free surface with a single layer of ciliated columnar epithelium.

As already indicated, this structure is very similar to that of the faucal tonsils, from which adenoids differ only in the absence of crypts, smaller proportion of connective tissue, and the presence of ciliated epithelium.

Symptomatology.—The symptoms and effects produced by adenoids are very varied. In a few cases when the growths are small there may be none, in others they may be slight; in the

majority they are well-marked and widespread, presenting a characteristic clinical picture. At first sight it might seem remarkable that an overgrowth of benign tissue resulting in such comparatively small masses could bring about serious results. The following considerations help to explain the incidence of symptoms which might otherwise appear incompatible with an apparently insignificant cause.

Situation.—The nasopharynx is a part of the main respiratory air-way. Any growth of tissue here is very liable to block up the posterior nares and cause nasal obstruction. Nasal obstruction induces “mouth-breathing.” In this way the important functions of the nose as a respiratory passage are lost, and the inspired air is unwarmed, unmoistened, and unfiltered.

The Eustachian tubes, soft palate, and pharynx with its apertures leading to larynx, lungs, and stomach, are all in immediate relation with the nasopharynx. Can we wonder that adenoids are frequently responsible for interference with the due performance of such functions as hearing, speech, and digestion?

Age-incidence.—As already pointed out, adenoids appear and persist during infancy and childhood when growth of organs and function is most active. Any interference with development at this time is obviously liable to bring about grave and permanent damage.

In any well-marked case of adenoids the hypertrophied lymphoid tissue encroaches to such an extent on the limited space of the posterior nares as to cause nasal obstruction. The patient is unable to breathe with comfort through the nasal passages, and unconsciously adopts the mouth as the only alternative portal of entrance for inspired air. The respiratory current is thus “short circuited” and the child becomes a “mouth-breather.” The symptoms and signs of mouth-breathing are characteristic, and are those for which in most cases advice is sought. The child’s nose is “stuffy,” or an observant mother notices that one or both nostrils seem often “blocked-up.” This condition may at first be intermittent, and the child is said to suffer from “repeated colds.” Respiration is often more or less noisy, and snoring when asleep is the rule. There is a constant tendency to “snuffling” or “hawking,” or the patient is conscious of “something at the back of the throat” setting up frequent, but always ineffectual, efforts at its removal. A peculiar harsh, dry, “barking” cough, for which no cause is found in the lungs, is common, and the patient is said to suffer from that mysterious affection “stomach-cough.” There

may be irregular discharge from one or other nostril; more frequently the child is noticed never to use a handkerchief. Dribbling of saliva during sleep often occurs, and the pillow may show slightly blood-stained discharge in the morning. Night-terrors or nightmare disturb the child's rest. Recurring headache is apt to be troublesome. Dysphagia, especially of fluids, has been observed.

These symptoms are among the commoner complaints for which the physician's advice is sought; or it may be that the pallor, listlessness, and general "delicacy" of the patient are the source of anxiety. The facies of such cases is characteristic. The mouth is persistently half-open, and the lower jaw remaining thus "dropped" imparts a curiously long appearance to the face. The upper lip is retracted, exposing the upper incisor teeth more or less constantly to view. The naso-labial fold tends to become obliterated, and the inner canthus of the eye is drawn down so that the eyelids are said to "droop." Prolonged disuse of the anterior nasal apertures gives a typical "pinched-in" appearance to the tip of the nose, which contrasts strikingly with the broad, flat, undeveloped bridge. A dimple often forms between its superior and inferior lateral cartilages. The child is usually pale and unhealthy-looking, and the deafness so often present induces an air of inattention which adds to the dull and vacuous appearance of the patient. In long-standing cases the superior dental arch becomes narrowed, the palate is high and the teeth crowded.

The presence of the growths in the naso-pharynx interferes with its resonating functions, while movement of the soft palate is impeded. In consequence the voice acquires a "dead" quality and a nasal twang: m, n, ng, are pronounced b, d, gg; thus "common" becomes "cobbod," "man" becomes "bad," and "sing" "sigg."

Deafness is a common and serious symptom, and may be brought about directly by obstruction of the Eustachian orifices, or indirectly by extension of coincident catarrh to the mucous membrane, with consequent absorption of air in the middle ear which is not adequately renewed. This deafness varies in amount and from day to day.

The entrance of air thus unwarmed, unmoistened, and unfiltered gives rise to recurring attacks of a catarrhal condition of larynx, trachea, or bronchi.

As a further result, serious and permanent thoracic deformity, such as "pigeon-breast," etc., may be produced in children, whose yielding ribs are prone to follow the falling-in of the chest-wall from pulmonary collapse thus induced.

Various "reflex" conditions have been described as due to adenoids, and certainly in many cases removal of these growths has been followed by a cure. They include such affections as paroxysmal sneezing, hay fever, spasmodic chorea, habit-spasm, asthma, epilepsy, torticollis, nocturnal enuresis, and stammering.

In some cases, as the result of these manifold symptoms and tendencies, the general vitality of the child is seriously impaired. Disordered sleep and repeated catarrh react on defective development of mind and body: the subjects are "backward" and "stupid"; they are always ailing, and pass from one childish complaint to another. Play and lessons alike have but transient charms, and in spite of the most fashionable "tonics" and frequent change of air the child remains "delicate."

On examining the fauces the tonsils are often found greatly hypertrophied and the soft palate is seen to be pushed forwards. Usually a viscid muco-purulent secretion is seen passing down the posterior pharyngeal wall from the nasopharynx. Occasionally, if the velum be lifted, the adenoids may be seen *in situ*. The growths may be examined by rhinoscopic inspection or by palpation. Even in children posterior rhinoscopy is often practicable, and may be conclusive, but digital exploration should be employed in all doubtful cases.

Standing on the seated patient's right side, the left arm of the physician holds the child's head against the left side, while his left forefinger pushes inwards the left cheek between the teeth, thus preventing any risk of being bitten.

The physician's right forefinger is now hooked round the soft palate and the whole of the nasopharynx can be thoroughly explored. The growths are felt as soft, smooth, rounded masses, which impart to the examining finger a sensation fancifully compared to a bag of worms. The finger is often slightly bloodstained on withdrawal.

Diagnosis.—The facial aspect is often characteristic, and digital examination of the nasopharynx affords conclusive evidence in any doubtful case. Congenital syphilis in infants may present similar symptoms, but other constitutional signs of this disease are usually present, while digital examination will yield negative results.

It must be remembered that nasal obstruction from such causes as polypi, or hypertrophied turbinals, will cause identical symptoms, facies, etc.; but these are rare in children, and careful examination of the nose, etc., will prevent any mistake in older patients. Occasionally in adults malignant growths commencing in the

nasopharynx may give rise to difficulty in diagnosis. Microscopical examination of a portion removed by operation should be resorted to in such cases.

Prognosis.—Adenoids themselves threaten no direct danger to life. But their presence is a very definite menace to such important functions as hearing and speech; while in children especially, development both of body and mind will almost certainly be retarded. It is true that in many cases the growths atrophy after the age of 25; but this is not constant, and is of comparatively little moment, since if left alone till then serious and permanent damage in the directions already indicated may have been sustained.

When once thoroughly removed they do not tend to recur. Operation is usually followed by the most happy results in general health; the sallow, dull child becomes bright and intelligent, the tendency to constant slight ailments disappears like magic, and development in all directions proceeds apace. It must be remembered that such brilliant results stand in direct relation to the patients' age, and the length of time the obstruction has existed; hence it follows that operation should not be postponed on account of the patients' youth, nor must the prognosis be too sanguine in long-standing cases.

Treatment.—The treatment of adenoids consists in their removal. Internal medication, nose or throat syringing, and the application of local remedies such as caustics, are useless and can only modify symptoms. To wait for serious symptoms is to court disaster.

The operation itself is simple and in capable hands practically devoid of risk. A general anæsthetic is always advisable in order that their removal may be thoroughly and systematically carried out. The operation is quite possible under cocaine, and some authorities use no anæsthetic, either local or general: under such conditions, in addition to the infliction of needless pain, the removal is apt to be incomplete, and the results consequently unsatisfactory.

The usual preliminaries to operation, such as abstinence from food, aperient, etc., must be carefully observed. Chloroform is preferable to ether, which tends to increase bleeding, and promotes an excessive secretion of mucus in the mouth. The anæsthetic should be cautiously administered until the conjunctivæ are insensitive, but stopping short of the point at which the laryngeal reflex (always the last to go) is inhibited. Usually no further

anæsthetic is required after the operation has once commenced. Most operators prefer to have the patient lying on the back with the head well extended hanging slightly over the edge of the table, with a small sandbag placed beneath the neck. In this position it is practically impossible for any blood to find its way into the larynx. A gag is inserted in the left side of the mouth, and the operator stands on the patient's right side.

A considerable variety of instruments has been devised for the removal of adenoids, of which Gottstein's curette and Loewenberg's forceps are those in most common use. The choice of these and the exact method of operating vary with different authorities. After removal the mucous membrane should be left quite smooth. Hæmorrhage is usually smart, but ceases spontaneously. The faucal tonsils if hypertrophied should be removed at the same time. The after-treatment consists in keeping the patient in bed for the first 24 to 36 hours, during which time his food should comprise only cold liquids. Ice to suck relieves any feeling of soreness. No local treatment is advisable. Complications are rare. Secondary hæmorrhage has occasionally occurred, and septic troubles such as otitis media or septic pneumonia may follow, but are very uncommon. It is probable that the removal of adenoids and the consequent wide area of absorbent lymphoid tissue left bare may render the patient for the time being unduly susceptible to such affections as tubercle, tonsillitis, measles, diphtheria, etc. Special care should be taken, therefore, to protect these cases so far as possible from such influences for at least two weeks after removal.

When the patient has recovered from the operation, it is important to remember that he should practise breathing through the nose with closed mouth: in other words, he has to unlearn a physiologically vicious habit. In the case of a child who has become a confirmed mouth-breather, regular exercises should be planned and carried out daily by an intelligent nurse, and it may be advisable to apply some simple contrivance for keeping the mouth closed during sleep.

HAY FEVER

SYN.: HAY ASTHMA, AUTUMNAL CATARRH, ROSE COLD

A disorder affecting the upper air-passages, manifesting itself as paroxysmal attacks of nasal catarrh, which are characterised by

marked periodicity, recur at a particular season, and, after persisting for a period of from one to three months, disappear, only to return at a corresponding time the next year.

Etiology and Pathology.—Hay fever is a disease of which much has been written and more yet remains to be elucidated. As its name implies, it was originally believed to be due to the irritation of the nasal mucous membrane by the pollen grains of certain plants. It is now established that this is only one factor in its complex etiology. Hay fever mainly affects the educated classes, and is especially common among the Anglo-Saxon, French, and German peoples. Men are more liable to it than women, and those who dwell in cities rather than the inhabitants of rural districts. Its manifestations usually first appear between the ages of ten and twenty, and recur each year in the summer months from June to September. A history of the disease in various generations of the same family may often be elicited.

Apart from these well-established facts, the exact nature of hay fever has been the subject of considerable controversy. It must be regarded as the resultant of three main factors:—

1. A neurotic constitution.
2. An abnormal condition of the nasal mucous membrane.
3. The presence of some directly exciting stimulus.

The relative parts played by these have given rise to differing opinions. Some authorities have considered hay fever a local disorder, while others held the neurotic element to be the main feature in its production.

To consider these factors briefly in detail:—

1. *The neurotic constitution.*—Under this heading it is to be noted that hay fever is mainly a disorder of civilised peoples, among whom we may expect a highly sensitive nervous organisation, and that it is especially common among the dwellers in cities.

Neurasthenics form no inconsiderable portion of its victims, and uneducated people are rarely attacked. Heredity shows a marked influence, and in 40 cases recorded by Sajous (quoted by Professor Shurley) 35 per cent had near relatives with a clear history of hay fever, while 42 per cent had asthmatic relatives. Other investigators have conclusively shown that either the disease itself or various nervous disorders (such as epilepsy, chorea, etc.) have existed to a greater or less degree in the families of most of the subjects of hay fever. The neurotic element was strikingly shown by the experiment in which an attack was induced in a susceptible subject by the sight of an artificial rose.

2. *Abnormalities in the nasal mucous membrane.*—Dr. Daly of Pittsburg first drew attention to the frequent incidence of nasal disease in cases of hay fever. The commoner abnormalities include polypi, adenoids, spurs or turbinal irregularities, hypertrophic rhinitis, and hyperæsthetic patches. These conditions exist so commonly among the subjects of hay fever as to suggest a causal relation, and many cases have been recorded in which the removal of the local disease has been followed by a cure. On the other hand, identical nasal lesions are common among those who never suffer from hay asthma, and it is possible that in long-standing cases the local disease may be in part at least a result of the symptoms.

3. *The stimulus.*—It has long been known that the onset of hay fever coincides with the beginning of the hay season, and that an attack is often precipitated by proximity to a hay-field. In 1873, Dr. Blackley of Manchester made a careful series of experiments, which showed that the pollen of certain flowering grasses diffused in the air acted as a direct excitant of hay fever, by their presence on and consequent irritation of the nasal or conjunctival membrane. Other stimuli, such as various kinds of dust, certain perfumes, emanations from animals, notably cats, may give rise to the attacks in susceptible individuals. Removal from the source of these stimuli often secures amelioration or immunity from the paroxysms. It is quite obvious that hay fever cannot be regarded as due to any one of these causes alone. It is hardly necessary to point out that only a small proportion of the host of neurotics are the victims of hay fever; that many patients are the subjects of nasal polypi, etc., who are quite free from such symptoms; while, lastly, every one is subject to the irritation of the pollen grains in the atmosphere, while but few suffer from hay fever. It would seem that a combination of factors is required for its production. The neurotic constitution acts as a predisposing cause, while the pollen grains or other stimuli serve as the exciting cause, whose effect is exaggerated by the diseased nasal lining. The symptoms are brought about by a sudden swelling of the abnormal mucous membrane due to vascular dilatation consequent on vasomotor paralysis. Hay fever presents intimate relations and analogies with bronchial asthma. Subjects of the one are prone to develop the other, and nasal abnormalities are common in both. It is probable that a similar combination of causes underlies the pathology of each, and that in hay fever the nasal mucous membrane, while in bronchial asthma the bronchial mucosa, is the seat of the vascular dilatation which sets up the symptoms in both.

Morbid Anatomy.—As already stated, the nasal mucous membrane usually shows definite abnormality. There is no special form of nasal disease peculiar to hay fever, and practically any form of nasal trouble may be found in its subjects. Chronic rhinitis in its varying forms, polypi, septal irregularities, and adenoids are common. Curiously hypersensitive areas of the mucous membrane are often present, the mere touching of which by a probe sets up violent sneezing. During the paroxysms there is much swelling and engorgement of the mucous membrane of the inferior and middle turbinate bodies. This is accompanied by free exudation of serous discharge.

Symptoms.—Hay fever in its typical form consists in a series of recurring paroxysms of nasal coryza. The attack usually commences somewhat suddenly with the symptoms of a severe cold in the head. There is a feeling of intense irritation in the nose, with much "stiffness." Sneezing is a marked feature, and its incessant repetition often forms the most distressing symptom in the case. Conjunctival inflammation and lachrymation are usually present in more or less degree. All the signs of nasal obstruction may be marked, and smell and taste are often interfered with, and there is a profuse watery discharge from the nose. Itching and discomfort may be referred to the palate or fauces, and the wearisome symptoms render the patient depressed and low-spirited. There is frontal headache, but little or no rise of temperature. The duration and severity of the attacks vary, and remissions of the general and local symptoms occur from time to time. The paroxysms recur with more or less severity during the period from June to August or September, and often terminate quite abruptly. Typical attacks of bronchial asthma may accompany or replace them. Examination of the nose during the height of an attack shows much swelling of the turbinate bodies, so that the nasal passages are often quite occluded.

Prognosis.—Hay fever does not *per se* threaten danger to life, and the general health is rarely seriously impaired. On the other hand, its persistent recurrence, the annoying discomfort produced, and its intimate relations with spasmodic asthma, sufficiently indicate the serious interference with the general well-being and usefulness of the individual. Local treatment directed to the nasal affection offers the best chance of cure. Hence it follows that the prognosis as regards cure is best in those cases with marked nasal abnormality, while the outlook is less hopeful when the neurotic element is marked. A proportion of the cases tends to become

chronic in spite of treatment, and recent observations seem to show that with advancing years many of these become the subjects of bronchial asthma.

Treatment.—There is no specific remedy for hay fever. Its rational treatment depends upon a knowledge of its etiological factors, and the application of measures suitable to each. The treatment must therefore be considered under the following:—

1. Treatment of the neurotic element.
2. Treatment directed to the exciting stimulus.
3. Radical treatment of the local nasal disease.
4. Palliative treatment.

1. *Treatment of the neurotic factor.*—This includes all those measures which conduce to improvement of the general health and make for stability of mind and body alike. The life and associations of the patient must be reviewed and unhygienic habits corrected. A wholesome dietary, open-air exercise, the morning cold “tub,” regular living, and avoidance of all forms of over-stress and excess must be enjoined. Dyspeptic and gouty tendencies demand special attention. Alcohol is best avoided. A course of such remedies as arsenic, strychnine, or phosphorus may sometimes be of use as tending to promote the stability of the nervous system. In other cases valerianate of zinc, asafoetida, or belladonna are valuable. Bosworth speaks highly of a combination of phosphide of zinc and belladonna:

℞ Zinci phosphid. grs. $\frac{1}{5}$.
Ext. bellad. grs. $\frac{1}{4}$.
M. ft. pil. i.

To be taken thrice daily after meals.

All these measures must be viewed as attempts at prophylaxis, and should be carried out when possible some weeks before the expected onset of the attacks.

2. *Treatment directed to the stimulus.*—The obvious indication under this heading is to remove the patient so far as is possible from the exciting cause of the attacks. Residence in agricultural districts usually aggravates the symptoms. Many patients remain free from attacks at the seaside, and others enjoy immunity only in the crowded districts of large cities. A sea-voyage or dry mountain air is sometimes the only means of relief. A patient who is compelled to remain in the country should protect himself by wearing a veil or coloured glasses. In the less common cases in which pollen grains are not the exciting cause, similar common-sense measures must be taken to avoid contact with the stimuli.

3. *Treatment of the local nasal disease.*—In every case of hay fever the nasal passages must be carefully examined, and any gross abnormality calls for active treatment. Spurs, polypi, adenoids, or hypertrophied turbinals should be removed. Hypertrophied mucous membrane or hyperæsthetic patches should be treated by the application of the galvano-cautery, or such caustics as chromic or trichloroacetic acid.

These various measures aim at destroying the hypersensitiveness of the nerve terminals, and thus to prevent the swelling of the "erectile" tissue. Such operative treatment should if possible be carried out some weeks before the expected onset of the attack. While their adoption promises the best hope of relief, it should be remembered that a guarantee of cure can never be given.

4. *Palliative treatment.*—As in other chronic diseases of complex etiology, multitudinous remedies have been advocated to relieve the symptoms of hay fever. Internal medication with such drugs as stramonium, antipyrin, the bromides, belladonna, and opium; inhalation of benzoin or chloroform; sprays of menthol or cocaine in oleaginous solution; injections of such antiseptics as carbolic acid, thymol, or quinine; snuffs or ointments containing morphine, atropine, bismuth, and the like: all these and many more have been in turn warmly advocated, and used as palliative measures.

Cocaine locally applied in 5% solution undoubtedly affords relief by causing contraction of the vessels. But its effect soon passes off, and the application must be renewed, while its frequent use too often leaves the sufferer worse than at first. This objection does not appear to apply to eucaine. Moreover, it must be remembered that the subjects of hay fever are many of them neurotics, and that such fall an easy prey to the cocaine habit. Comparatively recently suprarenal gland has been brought into use, by virtue of its action as a powerful local vaso-constricting agent. Applied locally, as a spray of adrenalin chloride (1 in 1000) once or twice daily, it produces all the excellent results of cocaine without its disadvantages.

Finally, in summing up the treatment of hay fever it must be remembered that each case must be considered on its own merits. The recognition and removal of gross nasal abnormality is all-important. Neurotic tendencies must be inquired for and combated. Careful observation will usually suggest the particular exciting cause in each case, and removal so far as is practicable

from its influence should be counselled. Suprarenal gland promises to be our most potent palliative ally.

SOME SPECIAL AFFECTIONS OF THE NASAL MUCOUS MEMBRANE

The development of the special study of the nose and nasopharynx has tended to show the importance of disease in these regions from the point of view of a complete diagnosis, to which indeed they sometimes afford a valuable clue. Thus it is that an examination of the nose may often form a necessary part of the routine investigation of a patient. It is proposed here to briefly outline a few of the more important local manifestations of general diseases occurring in the nasal mucous membrane, whether these be the sole evidence, or a part of a more widespread distribution.

CHRONIC NASAL CATARRH.—A purely local affection of the nose, common in children and adults, demanding mention here on account of its frequent association with catarrh of other parts of the respiratory tract. A great variety of factors, both constitutional and local, are believed to be concerned in its etiology. The mucous membrane presents all the changes of the various stages of chronic inflammation. Usually there are no constitutional symptoms. Varieties of the disease are described, *e.g.* hypertrophic, atrophic, fibrinous, membranous, etc. The constant symptom is a persisting discharge, usually mucopurulent and often offensive. Mouth-breathing with all its results is common. For fuller details special works on the subject must be consulted.

SPECIFIC FEVERS.—In various specific fevers the nasal lining is often implicated, but usually only as a part of the general manifestation.

a. Influenza.—Symptoms of acute rhinitis are common during the first forty-eight hours of an attack of catarrhal influenza. As a rule they subside within three days, but occasionally persist for as long as a fortnight. In the majority of cases the nasal trouble is insignificant, but Ewald regards influenzal rhinitis as a common cause of sinusitis, and Weichselbaum has reported abscess of the antrum and frontal sinus in sixteen fatal cases of influenza.

b. Measles.—The earliest symptom is usually a "running" from nose and eyes. The signs of acute rhinitis persist as a rule till the appearance of the eruption on the fourth day, when they are often at their worst. Nasal complications are rare.

c. Roetheln.—In German measles a slight nasal catarrh is a common early symptom.

d. Variola.—Occasionally in cases of smallpox a purulent rhinitis may add to the patient's suffering, and the characteristic eruption is sometimes seen on the nasal mucous membrane.

e. Scarlet fever.—In severe attacks of scarlet fever, especially those accompanied with much ulceration of the fauces, the septic process may extend to the nasal passages. In the worst cases there may even be sloughing of the nasal lining.

f. Diphtheria.—Diphtheria may attack the nose primarily, or the nasal mucous membrane may be implicated by extension from the pharynx. In either case all the constitutional symptoms of the disease are present (see Vol. I. p. 163). The nasal affection is evidenced by the presence of a discharge from the anterior nares which is thin and mucopurulent, often bloodstained, and frequently causes excoriation of the upper lip. Children are most often the subjects of nasal complication. The typical diphtheritic membrane is found in the local lesions, and the Klebs-Löffler bacillus is always present, usually in association with other septic micro-organisms. The lymphatic glands are always inflamed. There is as a rule marked nasal obstruction, and the diphtheritic process may involve nasopharynx, Eustachian tubes, etc. The presence of such a nasal discharge in a sick child should always suggest the possibility of diphtheria: probably many cases are undetected. The constitutional symptoms in nasal diphtheria are often very severe. It must be remembered that any of the important complications or sequelæ of the disease (nephritis, peripheral neuritis, etc.) may follow.

NASAL GONORRHOEA.—The nasal mucous membrane of newly born infants is occasionally infected by the gonococcus during delivery. The symptoms and signs are those of an acute rhinitis with much swelling and thick yellow discharge. The conjunctivæ are often simultaneously affected.

NASAL TUBERCULOSIS.—Recent investigation has shown nasal tuberculosis to occur more commonly than was formerly supposed. It is usually secondary to tuberculosis of lungs, or other parts, but Herzog has reported twenty cases of primary nasal tubercle. The local manifestation occurs either as an ulceration or as a small granulomatous tumour. The ulcers may be single or multiple, are found most commonly on the septum, and show the usual characters of tuberculous ulcers in other parts of the body. The tumour formation is seen more often on the turbinals, resembling a small

papilloma. Tubercle bacilli have been found in scrapings or sections from either form. The nasal symptoms are as a rule quite insignificant, and in the majority of cases the disease tends to remain localised. It is by no means improbable that the nose may be the primary focus of tuberculosis more commonly than is usually supposed. In a recent monograph Dr. Knowles Renshaw of Manchester has published experiments in which he showed the readiness with which the nasal mucous membrane became infected in guinea-pigs.

NASAL SYPHILIS.—The nose may be affected in either congenital or acquired syphilis.

(a) *Congenital syphilis*.—A catarrhal rhinitis is usually the earliest symptom of congenital syphilis, and is often present a few days after birth. There is swelling of the nasal mucous membrane, and an irregular muco-purulent discharge. Nasal obstruction is a marked feature, and in consequence the infant takes the breast with difficulty. The term “snuffles” fittingly describes the impediment to nasal respiration. Other constitutional signs of syphilis, such as skin eruptions, mucous tubercles, etc., together with the characteristic malnutrition and the wizened “old man” appearance, render the diagnosis easy. It should, however, be remembered that infants may suffer from rhinitis due to other causes, and a history of “snuffles” alone is not sufficient ground for a diagnosis of inherited syphilis. In untreated cases ulceration of the mucous membrane may follow and involve cartilage and bone, producing serious and permanent deformity.

(b) *Acquired syphilis*.—Primary chancre of the nasal apertures has been recorded, but is very rare. Secondary lesions, such as mucous patches, occasionally occur; but they are not common, and differ in no way from those found on other mucous membranes. Tertiary syphilis of the nose is frequently seen; gummata, ulcers, and necrosis of bone or cartilage are all of common occurrence. Gummata are most often seen on the septum: they are usually unilateral, and appear as smooth, rounded tumours of reddish hue. Pain and symptoms of nasal obstruction may be complained of. A gumma may persist for months, or may rapidly break down, leaving deep ulceration. Tertiary syphilitic ulceration of the nose as a rule involves bone or cartilage or both. Extensive areas of bone are often involved and destroyed, and much deformity results. The discharge is copious, thin, and may be very offensive, and the usual characteristics of bone suppuration are present. Evidences of syphilis in other parts of the body will be found.

GLANDERS.—In acute glanders the nose may be affected either primarily or secondarily. This disease is rarely seen in man, and a description of its symptoms is given in Vol. I. p. 218. Nasal implication is shown by swelling and tumefaction of the mucous membrane, and the presence of the characteristic nodules. Later these break down, leading to irregular deep ulceration. There is a constant nasal discharge, at first thin, but becoming purulent, sanious, and very offensive.

All the constitutional signs of the disease are present.

Treatment.—The treatment of acute coryza and hay fever is detailed in the special articles on these diseases. Chronic nasal catarrh calls for local treatment, for the details of which special works on nasal surgery should be consulted. The nasal implications of the specific fevers rarely demand local treatment; but in the severe forms of inflammation attended with destruction of tissue seen in septic scarlet fever, frequent syringing with a warm solution of freshly made chlorine gives the best results.

R Potass. chlor. \mathfrak{v} iss.
 Ac. hydrochlor. pur. \mathfrak{v} i.
 Glycerin \mathfrak{v} iii.
 Aq. ad \mathfrak{v} xii.

Add the hydrochloric acid to the powdered chlorate in a dry bottle, and dissolve the evolved chlorine by adding the water little by little, with frequent shaking.

This solution, mixed with an equal part of hot water, should be syringed through the nostrils, as described in Vol. I. p. 290.

In nasal diphtheria the same remedy will be found useful: antitoxin injections and the appropriate constitutional treatment must of course be adopted. Nasal gonorrhœa must be combated by the usual antiseptic applications. In nasal tuberculosis removal or destruction of the affected area by the knife, cautery, or scraping should be carried out when practicable. Otherwise the treatment differs in no way from the treatment of tuberculous ulcers elsewhere. Nasal syphilis calls for the exhibition of mercury and iodide of potassium. There is no specific treatment for nasal glanders.

LEWIS SMITH.

DISEASES OF THE LOWER RESPIRATORY TRACT

Diseases of the lower respiratory tract are of special importance on account of their wide prevalence at all ages and the large mortality to which they give rise. They occur in very varying degrees of severity, and may either run a short and acute course or continue in a chronic form for months or years, sometimes remaining to the end of life. Of the former class, some, such as the milder cases of bronchial catarrh, may be apparently very trivial affections, while others, such as acute pneumonia, more often than not are serious and severe. From the statistics of hospitals it would appear that, without taking account of pulmonary tuberculosis, 30 per cent of all the graver cases of illness coming under the physician's care are affections of the respiratory organs. From the reports of the Registrar-General we find that if we exclude pulmonary tuberculosis, about 20 per cent, and if we include it, about 28.5 per cent of the deaths from all causes and at all ages are due to diseases of the respiratory tract. The mortality from respiratory disorders is heaviest at the two extremes of life, and, except between the ages of ten and fifteen, is considerably greater among males than among females.

GENERAL ETIOLOGY

The character of disorders of the lower respiratory tract is affected to a considerable degree by age and sex. Certain influences, such as heredity, constitution, obstruction of the upper air-passages, deformities of the chest-wall, occupation, habits, and climate, have an indirect action in the production of chest and throat affections. Some agencies, such as dust and other impurities in the air, injury, foreign bodies, and micro-organisms, have a direct causal relation. Others, again, such as alcohol, cold and chill, and the effects of various diseases, have probably both a direct and indirect action.

AGE.—The lower respiratory tract is most susceptible to morbid influences at the two extremes of life. The very young and the old are peculiarly liable to pneumonia, and to bronchitis, which not infrequently extends to the bronchioles and alveoli, with resulting broncho-pneumonia. After the period of infancy the child shows

greater resistance, and bronchitis and pneumonia, though not uncommon, are less serious affections. Asthma frequently occurs for the first time under the age of ten, but seldom between ten and twenty. Emphysema may occur in early childhood, but is then usually secondary to collapse and bronchitis. During adolescence both bronchitis and pneumonia are relatively less frequent than at a later period. They are met with in about the same proportion at all ages during adult life, becoming more frequent again in old age. Chronic affections, such as chronic bronchitis and laryngitis, are uncommon except in adults, and chronic bronchitis and emphysema are most frequent after forty-five. Pulmonary phthisis is comparatively rare before the age of fourteen, and is most common between the ages of twenty and forty-five, but may be met with at any age. New growths of the lungs most frequently develop during and after middle life.

SEX.—In childhood sex appears to have little influence on the incidence of pulmonary disease; but among adults, probably on account of the differences in habits and occupation, the male sex suffers more than the female. Men suffer from all chronic affections of the larynx, from phthisis, and from pneumonia, to a considerably larger extent than women. As regards bronchitis the sexes suffer fairly equally until thirty-five, after which age males prove somewhat more liable than females. Males suffer about twice as frequently as females from emphysema. The same holds true for asthma, except between the ages of twenty and forty, when females show an equal liability.

HEREDITY.—In certain families there appears to be a special tendency to catarrhal affections, and bronchitis shows itself in successive generations. Asthma also runs in families, and often occurs in association with nerve disorders, such as hysteria, epilepsy, or chorea, in parents or collaterals. In emphysema, too, a hereditary tendency has been frequently noted. In these affections some evidence of heredity may be traced in about 40 per cent of the cases, if one includes the affection of collaterals as well as that of the parents. Several children of the collaterals in a family may be affected without the parents having suffered. It is probable that there is some inherent weakness of the mucous membrane or elastic tissue intermingled with hereditary delicacy of constitution.

In the case of pneumonia, also, a hereditary influence probably exists. But it is especially in relation to pulmonary tuberculosis that the most marked hereditary influence has been observed. In nearly thirty per cent of the cases of phthisis it is observed that one

or both parents have died of the disease, and in another twenty per cent the disease is found to have occurred in some other member of the family. The experience of life-assurance offices, moreover, shows that there is a higher than the average rate of mortality from phthisis among persons with a family history of the disease. The influence of heredity shows itself in a larger proportion of cases occurring before the age of twenty-five than after. It has been observed that the disease assumes a more severe form in cases with a family history of tubercle than in others. The hereditary tendency appears to be more marked in the case of females than in males, and some have believed that the maternal influence was stronger than the paternal, but this is not borne out by statistics. There may be an inherited tendency to the disease, as shown by its appearance in several collateral members of the family, although the parents themselves have not been affected, and this has been attributed to consanguinity, disparity of age, or too early marriage of the parents. Tubercle in the newly born is so rare as to be a pathological curiosity, and in practically all cases of pulmonary tuberculosis the disease is acquired after birth. What is inherited is not the disease itself but a greater susceptibility to it, and it may be pointed out that the person who has phthisical relatives runs an increased risk of personal infection.

CONSTITUTION.—All respiratory diseases are peculiarly liable to attack the less robust, and this is specially the case with those diseases which depend on microbic infection, such as pneumonia, broncho-pneumonia, and phthisis, although these often attack apparently healthy persons. Badly nourished infants frequently fall victims to bronchitis and broncho-pneumonia. Children and adults who have undergone hardships and privation, and women weakened by prolonged lactation and repeated child-bearing, are specially liable to pneumonia and phthisis if exposed to the exciting causes of these diseases.

Persons who are under the average weight for height suffer to a greater extent from phthisis than those who are of normal proportions or over weight. Obesity, however, has its disadvantages in other directions, and fat middle-aged persons are specially liable to bronchitis.

From the time of Hippocrates a certain special type of constitution has been described as associated with phthisis, the chief characteristics being a clear complexion, delicate skin, fine silky hair, tapering fingers, and precocious intellect ; and experience shows that persons presenting such characteristics are more vulnerable than others. Persons with blue eyes, reddish hair, and fair skin have

been considered specially susceptible, but the evidence of this is inconclusive.

MECHANICAL IMPEDIMENTS.—Obstruction to the upper air-passages, whether by nasal polypi, hypertrophy of the turbinate bones, adenoid vegetations, or enlarged tonsils, by interfering with the free entrance of air into the lungs, or by leading to mouth-breathing, predisposes to chronic laryngitis, chronic bronchitis, asthma, and emphysema.

Deformities of the chest-wall, such as those resulting from rickets or spinal curvature, by interfering with the free expansion of the lungs, predispose to bronchitis, emphysema, and phthisis.

MODE OF LIFE.—*Occupation.*—All occupations carried on in over-heated, over-crowded, and badly ventilated rooms, or in a dusty atmosphere, predispose to bronchitis, pneumonia, and phthisis. Occupations involving exposure to cold and wet also contribute to the causation of bronchitis and pneumonia. Among such unhealthy occupations may be mentioned those of hotel-servants, dock-labourers, coal-heavers, employés in iron and steel and all dust-producing factories. Occupations involving forced expiratory efforts, such as glass-blowing and playing on wind instruments, or requiring severe muscular effort and the lifting of heavy weights, favour the production of emphysema.

Alcoholism.—It is difficult to separate the influence of occupation from the effect of habits. Alcoholism, by undermining the constitution, diminishes the resistance to the tubercle bacillus and the pneumococcus. Pulmonary phthisis and pneumonia are accordingly found to be not infrequent among heavy drinkers.

Smoking in excess, especially cigarette smoking, helps to set up chronic laryngitis and chronic cough, and may indirectly lead to bronchitis and emphysema.

CHARACTERS OF THE AIR BREATHED.—*Dust.*—The inhalation of dust is irritating to the respiratory tract, and when continued is apt to lead to chronic bronchitis, emphysema, and chronic phthisis with chiefly indurative changes in the lungs. A special name has been given to the induced pulmonary condition “pneumono-koniosis,” and the terms anthracosis, chalicosis, and siderosis have been suggested for the different forms set up by coal, stone, and metal dust respectively. Coal-miners and metal-moulders are specially exposed to the effects of coal or charcoal dust; stone-masons, millstone-makers, diamond-polishers, stone- and glass-cutters, and potters to the dust of stone or sand; metal-workers, needle-grinders, etc., to metallic dust, especially oxide of iron; and among others whose occupations

are peculiarly dusty are mill-hands in cotton and shoddy factories, horse-hair beaters, straw-hat makers, grain-shovellers, and millers.

Organic and other impurities.—No less potent as a cause of pulmonary disease than the presence of dust is that of organic impurities in the air, especially human exhalations. The air of badly ventilated rooms and workshops where people are crowded together is highly injurious, and disposes to all forms of pulmonary disease. The inhalation of iodine, ipecacuanha, or ozone in excess has been observed to induce an attack of asthma or bronchitis in susceptible persons. Injury to the mucous membrane as the result of inhalation of powerful irritating vapours such as strong fuming nitric acid will set up acute bronchitis. Gangrene of the lung has been observed to follow the inhalation of such caustic vapours as those of sulphuric acid or picrate of potash.

FOREIGN BODIES.—When arrested in the larynx or trachea, foreign bodies give rise to inflammation or ulceration in the surrounding parts. When a foreign body lodges in a bronchus, it is generally on the right side, the right main bronchus being more nearly a continuation of the trachea than the left. When the body completely blocks a tube, collapse of the portion of lung supplied by that tube follows. When the obstruction is partial, then vesicular or interstitial emphysema is produced. Later changes which may arise are local inflammation, œdema, ulceration, and abscess, which may go on to gangrene or lead to perforation of pleura or rupture of vessels.

TRAUMA.—An injury such as a severe blow or bruise over the chest-wall is sometimes quickly followed by pleurisy or acute lobar pneumonia, and probably acts by directly preparing the way for the microbe. Direct injury to the lung, such as a gunshot-wound or a stab, sets up as a rule not acute pneumonia but a low form of inflammation which may terminate in gangrene. An injury may also prove the starting-point for pulmonary phthisis, as shown by cases recorded from time to time. A form of phthisis observed among boatmen on the Rhône has been attributed to chronic trauma from pressure on the chest-wall by the pole used for propelling the boat.

COLD AND CHILL.—In the popular mind there are no more potent causes of respiratory diseases than cold and chill. It is on account of this association that ordinary catarrhs are spoken of as "colds." A previous exposure to cold is often observed before an attack of bronchitis or pneumonia. The influence of cold and chill is probably in lowering the resisting power, and so permitting microbes present in the mouth or the air to make a successful invasion of the organs of respiration. Susceptibility to cold to a

large extent depends on the mode of life, and is greatly increased by living in close, over-heated, badly ventilated rooms.

THE INFLUENCE OF SEASONS. — Respiratory complaints are popularly associated with winter and spring, and the seasons at which they are most prevalent are those in which rapid changes of temperature are most common, and when cold winds and fogs abound. In winter and spring bronchitis prevails, while in April and May pneumonia is most frequent. When there are sudden changes of temperature, persons are less prepared for the cold, and accordingly suffer more from it. It has been suggested that the pneumococcus acquires special virulence in the spring, but this seems very doubtful.

CLIMATE. — Climates characterised by frequent and sudden changes of temperature are favourable to pulmonary diseases. Where cold winds prevail, and the climate is cold, damp, and changeable, bronchitis and phthisis are common. To a considerable extent these conditions act by inducing susceptible persons to remain indoors with closed windows, and in this way it is the impure atmosphere of the house which affects them, rather than the cold winds outside. Phthisis is common or rare according as the climatic conditions keep people indoors or permit of them leading an outdoor life. Pneumonia is peculiarly a disease of temperate and changeable climates. Phthisis and catarrhal affections have been observed to prevail where there is an excess of moisture in the subsoil, and have greatly diminished when the subsoil has been efficiently drained.

PREVIOUS DISEASES. — The influence of one attack of bronchitis is to predispose to another, and chronic bronchitis not infrequently follows repeated attacks of the acute form, favoured thereto by the emphysema which has been developed. In the case of pneumonia a similar tendency to repeated attacks (generally in the same lung) exists, but is less usual. As many as twenty-eight attacks of pneumonia have been observed in one individual. One pulmonary disease is apt to complicate another: broncho-pneumonia follows on bronchitis in infancy and old age, phthisis ensues on chronic bronchitis in adults, and asthma, emphysema, and bronchitis are frequently combined. Bronchiectasis and tumours of the lung not infrequently terminate in gangrene, which also occasionally supervenes on broncho-pneumonia or pneumonia. Burns of the skin, when extensive, may be complicated with broncho-pneumonia. Glanders, farcy, acute rheumatism, intermittent fever, puerperal fever, gangrene of the mouth, scurvy, and purpura may be followed

by pneumonia. Diabetes, syphilis, and congenital heart disease dispose to phthisis; and chronic gout, the degenerative changes of advancing years, renal disease, and chronic congestion from heart disease to bronchitis and pneumonia. The acute fevers, such as measles, scarlet fever, diphtheria, influenza, typhus, typhoid, erysipelas, and whooping-cough are not infrequently complicated with laryngitis, bronchitis, broncho-pneumonia, pleurisy, or pneumonia; and measles in particular is apt to be followed by phthisis. Certain diseases, such as smallpox, scarlet fever, mitral stenosis, malaria, gout, and cancer, have been thought to exercise an antagonistic action to phthisis. Whatever the explanation may be, it appears to be uncommon for mitral stenosis or gout to be associated with phthisis, but in the case of the other diseases antagonism is doubtful. In the case of cancer and phthisis the age periods at which the two diseases are prevalent are different, but in spite of this a combination of the two is not very uncommon.

INFECTIONS.—A number of diseases of the respiratory organs are associated with micro-organisms and strictly belong to the class of infective diseases. Thus acute pneumonia is found to be connected with a special micro-organism, and sometimes appears to take on the form of an epidemic disease (*see* Vol. I. p. 178). Among other infective pulmonary diseases are tuberculosis and actinomycosis.

(1) *The pneumococcus* discovered by Fränkel and Sternberg is a micrococcus which is found to be present in the exudate and in the sputum in a large number of cases of pneumonia, and not uncommonly also in broncho-pneumonia. The pneumococcus in preparations from sputum or the blood of inoculated animals is oval or shaped like a lance-head, but in cultures in liquid media the cells are spherical or nearly so. The cocci are usually united in pairs, or form short chains of three or four elements. A thin gelatinous capsule, when the specimen is obtained from sputum or exudates, encloses the cell. The microbe is non-motile, and while aerobic is a facultative anaerobe. It grows in a variety of culture media, but a slightly alkaline reaction of the medium is essential in the case of typical pathogenic forms. A temperature nearly that of the blood is most favourable for its growth, which will not take place at the ordinary room temperature. The microbe is highly pathogenic for mice and rabbits, and less so for guinea-pigs; and a culture injected into the lungs of rabbits sometimes produces a typical fibrinous pneumonia (*see* Vol. I. Plate I. and pp. 178-181).

The pneumococcus is present in the saliva of 20 per cent of healthy persons. It is not satisfactorily explained how it seems

at times to take on a special virulence, and sets up such serious illnesses, when it is so often an apparently harmless inhabitant of the mouth of healthy persons. The facts, however, that it is almost constantly found in the sputum and in the exudate in cases of pneumonia, and that it is found in the lesions of various complications of pneumonia, such as empyema, endocarditis, meningitis, acute abscesses, and arthritis, point to its being the exciting cause of the disease. The microbe is difficult to cultivate from the sputum or exudate; but if a susceptible animal, such as the mouse or rabbit, be inoculated with matter containing the microbe, the animal will die within a few days, and the coccus may be readily cultivated from the blood.

Another microbe has been described in the lesions of pneumonia. This is a very short capsulated bacillus (the bacillus of Friedländer). It has only been found in a small proportion of the cases, and cannot be considered as the cause of the disease.

(2) *The tubercle bacillus*, which has already been described in the article on tuberculosis, is the direct cause of the various forms of laryngeal and pulmonary tuberculosis. There are three modes in which the tubercle bacilli may gain access to the respiratory tract, namely, inhalation, ingestion, and inoculation. By means of inhalation the bacillus is carried directly to the part which it affects. Numerous experiments have shown conclusively that the inhalation of air in which is suspended dried tuberculous sputum will induce tuberculosis in even the most refractory animals. In the case of man it is by inhalation that the disease is most usually contracted, and we find that the lungs and bronchial glands are far more frequently affected with tubercle than any other parts of the body. A phthisical patient may, in the course of the day, expectorate millions of bacilli, and if he is careless in the disposal of his sputum must infect the place where he lives. It has been proved that dried sputum will retain its virulence for a long time. Experiments with dust, collected in various quarters of Berlin by Cornet, showed that nearly 33 per cent of the specimens were infective, the dust being most frequently virulent in hospital wards and in private rooms occupied by consumptive patients. Recent experiments made by Coates in Manchester show that in houses which have been occupied by careless consumptive invalids the dust is infective in more than 50 per cent of the cases. In no instance was the dust of even the dirtiest houses found to be infective when no case of tuberculosis had occurred in them during the previous three years.

When the bacillus is introduced into the body by ingestion, it may enter the blood current and so be carried to the lungs, but it is more likely to cause abdominal or general tuberculosis than the pulmonary form. It has been generally held that tuberculosis can be communicated to man from the lower animals, and especially from tuberculous cattle, and that, while milk is the principal source of danger, the disease may also be communicated by means of meat. Recently Professor Koch has thrown doubt on the infectiveness of bovine tubercle for man, having himself failed in his attempt to inoculate cattle with human tubercle. If, however, as is generally held, danger exists, it may be averted by thoroughly boiling the milk and thoroughly cooking the meat. Milk has been found to be only infective for the lower animals when the cow has tuberculous disease of the udder. Tubercle very rarely occupies the fleshy parts, and the chief danger in the case of meat is that it may be accidentally smeared with tuberculous matter from the internal organs.

Inoculation is a rare method of accidental infection in man, the hands being the usual seat of the primary lesion, which later may be followed by pulmonary disease. Cases of this kind are chiefly observed among washerwomen, post-mortem room porters, and demonstrators and workers in clinical or pathological laboratories.

It may be considered as one of the best-established facts in medical science that phthisis is conveyed from one person to others by means of the expectoration, and that this is the usual mode of infection. Neither the sweat nor the breath of an infected person contains bacilli. Both theory and experience show that if proper precautions are taken with regard to the disposal of the expectoration the chances of infection are reduced to a minimum. It has been suggested that little particles containing bacilli may be projected into the air during the act of coughing, but the risk of infection in this way is very small (*see* Vol. I. pp. 220-234).

(3) *Other micro-organisms* play an important part in the etiology of broncho-pneumonia, and in modifying the course of other pulmonary diseases, such as phthisis. The streptococcus pyogenes and the staphylococcus pyogenes aureus are found sometimes alone, sometimes together or along with the pneumococcus, in cases of broncho-pneumonia. In some cases of pneumonia of abnormal type the streptococcus appears to be the infective agent. In certain stages of phthisis, pneumococci, streptococci, or staphylococci are found in the sputa and in the lesions in association with active disease and breaking down of the lungs. The presence of these microbes is, however, a secondary and accidental

phenomenon. In putrid bronchitis a special bacillus has been observed by Lumnitzer and others.

(4) Certain infective diseases of the respiratory organs depending on micro-organisms have been already considered in the first volume of this work. We refer to the pneumonic type of plague (Vol. I. p. 133), the respiratory variety of anthrax known commonly as woolsorters' disease (Vol. I. p. 153), the affection of the larynx in leprosy (Vol. I. p. 240), pulmonary actinomycosis (Vol. I. p. 247), and aspergillar pneumomycosis (Vol. I. p. 250). Syphilis no doubt depends on a specific micro-organism, although this has not yet been discovered.

(5) Certain pulmonary diseases, as hydatids and distomiasis, are dependent on the invasion of the lungs by parasites.

MORBID ANATOMY AND GENERAL PATHOLOGY

INFLAMMATIONS

ACUTE LARYNGITIS. — Adults are more often attacked by this malady than children, and males more frequently than females. In the case of females there seems to be an increased liability to it at the menstrual periods. Although commonly ascribed to colds, it is seldom produced by that cause unless there has been previous exposure to the impure air of badly ventilated rooms or vehicles. Over-use of the voice, defective nasal respiration, chronic alcoholism, excessive smoking, the inhalation of dust or irritant vapours, gout, syphilis, and tuberculosis are among recognised causes. Acute laryngitis occurs also as a complication of influenza, smallpox, measles, and other specific fevers. Acute exacerbations are apt to occur in the course of chronic laryngitis. The signs of inflammation are usually most marked in the neighbourhood of the glottis. The cords are hyperæmic and injected, and slightly swollen from infiltration of the submucosa. In some cases slight superficial ulcerations have been observed in the situation of the processus vocales, the interarytenoid fold, or the anterior commissure. In certain inflammations of specific origin, such as erysipelas and tubercle, or when due to trauma, œdema may be a marked feature.

CHRONIC LARYNGITIS.—This condition is sometimes a sequel of acute catarrhal laryngitis. Over-use of the voice, excess in alcohol and tobacco, and other causes of the acute affection, largely contribute to its production. It is much more common in males than females.

Three forms of chronic laryngitis have been recognised: a hypertrophic form, an atrophic form, and a glandular form. The first is common, the other two comparatively rare. In the atrophic form, *laryngitis sicca*, the mucous membrane is atrophied and there is frequently associated chronic atrophic rhinitis. In the glandular form the special feature is that the minute racemose glands are enlarged and granular pharyngitis commonly co-exists. The hypertrophic or common form is characterised by congestion and thickening of the vocal cords, the interarytenoid fold, and the ventricular bands. The cords are reddened as well as thickened,

and are sometimes irregular in outline, but are seldom ulcerated. In some cases the chronic thickening is very marked, and a special name, *pachydermia laryngis*, has been applied to the condition. Sometimes the swelling is limited to the anterior extremities of the cords near the commissure, but more commonly it affects the posterior extremities, which are occupied by symmetrical, oval, pinkish-red growths. It is characteristic that there is usually a cup-shaped depression on one growth, into which fits a corresponding prominence on the other when the cords are in contact. The interarytenoid fold may be similarly affected, having a thickened, swollen, and corrugated appearance.

A variety of pachydermia, variously known as "chorditis tuberosa," "trachoma," "singers' node," or "teachers' node," consists of small poppy-seed-like, hard and consistent growths, seated on the upper surface or free border of one or both cords, about the junction of the anterior with the middle third, the membrane surrounding which is usually hyperæmic.

PERICHONDritis OF THE LARYNX.—Primary perichondritis is extremely rare, but it is a possible complication of catarrhal laryngitis. In the secondary form it results from syphilis, tuberculosis, and malignant disease, from the infectious fevers, especially enteric, from gout and diabetes, and from septic inflammation, trauma, scalds, and foreign bodies. It has been attributed to pressure of the cricoid on the bodies of the cervical vertebræ in the dorsal decubitus of old people, and to the frequent passage of œsophageal bougies. It chiefly affects the cricoid cartilage, especially its posterior surface, and the arytenoids. In the usual or suppurative form the perichondrium becomes thickened, and pus collects between it and the cartilage. The abscess ruptures into the larynx or surrounding parts, and the cartilage becomes carious and necrotic, and sooner or later is exfoliated, with resulting great deformity of the larynx. An adhesive form is also met with in which there is considerable thickening, but without suppuration.

The capsule and articular surfaces of the crico-arytenoid joint are frequently involved in inflammatory affections of the larynx, especially perichondritis, with resulting fixation or dislocation (luxation) of the arytenoid from the articular surface of the cricoid.

ACUTE TRACHEITIS AND BRONCHITIS.—Acute tracheitis and bronchitis is one of the most common diseases. The conditions which tend to produce it are those which also cause acute laryngitis, and have been already considered in detail in the section on general etiology. The malady is common to all ages and in

different cases is of very varying degree. It may be a very mild affection or one of the most serious nature, and while often primary is also very frequently secondary. The primary form closely resembles a specific disease; and although no microbe has been identified with the malady as the pneumococcus has been with pneumonia, a specific micro-organism may yet be found. Various micro-organisms such as streptococci and staphylococci have been found in the inflamed mucosa, especially when the bronchitis has arisen in specific fevers.

In a typical case of this affection the walls of the trachea and bronchi become hyperæmic and swollen, while secretion accumulates on their surface. Although the mucous membrane when inflamed is probably always hyperæmic during life, there may be no obvious signs of this after death. In some cases it is pale and in others red or purple, and it may be studded with punctiform hæmorrhages. Marked injection is usually limited to the larger tubes. In most cases, however, definite changes may be observed on microscopical examination. These consist of inflammatory hyperæmia of the inner coat or mucosa proper, oedema of the basement membrane, and swelling of the cells of the ciliated columnar epithelium, of which many are shed, while new cells are in process of formation by germination of the deeper layers. The mucosa, submucosa, the muscular layer, and adventitia are infiltrated with small cells, many of which have made their way into the interior of the tubes, and the mucous glands are swollen and distended with secretion. The mucus or mucopurulent matter lining the tubes resembles the expectoration, but contains a larger proportion of epithelial cells, a result perhaps of the continued shedding of these cells after death.

When the bronchitis extends to the smaller tubes, these not uncommonly become dilated, blocked with secretion, or obstructed by swelling. Oedema and congestion of the lungs are frequent secondary changes, and collapse and broncho-pneumonia as well as emphysema, either old or recent, are not uncommon. The bronchial glands are generally swollen and hyperæmic, and the right side of the heart is usually distended with blood.

In CHRONIC TRACHEITIS AND BRONCHITIS there is usually marked thickening of the mucous membrane, which is reddish or slaty-gray, and sometimes has a granular, velvety appearance due to the presence of granulations resembling small, firm, villous growths. The surface of the tubes is to a large extent denuded of columnar ciliated epithelium, which is replaced by small oval cells attached

to the basement membrane. There is obvious cellular infiltration in the muscular and fibrous layers, which, atrophied in some cases, may be hypertrophied in others, causing general fibroid thickening of the tubes. The mucous glands are not uncommonly atrophied, and the cartilages are thickened and in elderly subjects sometimes calcareous. The lumina of the larger tubes are commonly narrowed, while those of the smaller tubes are dilated. The bronchial glands

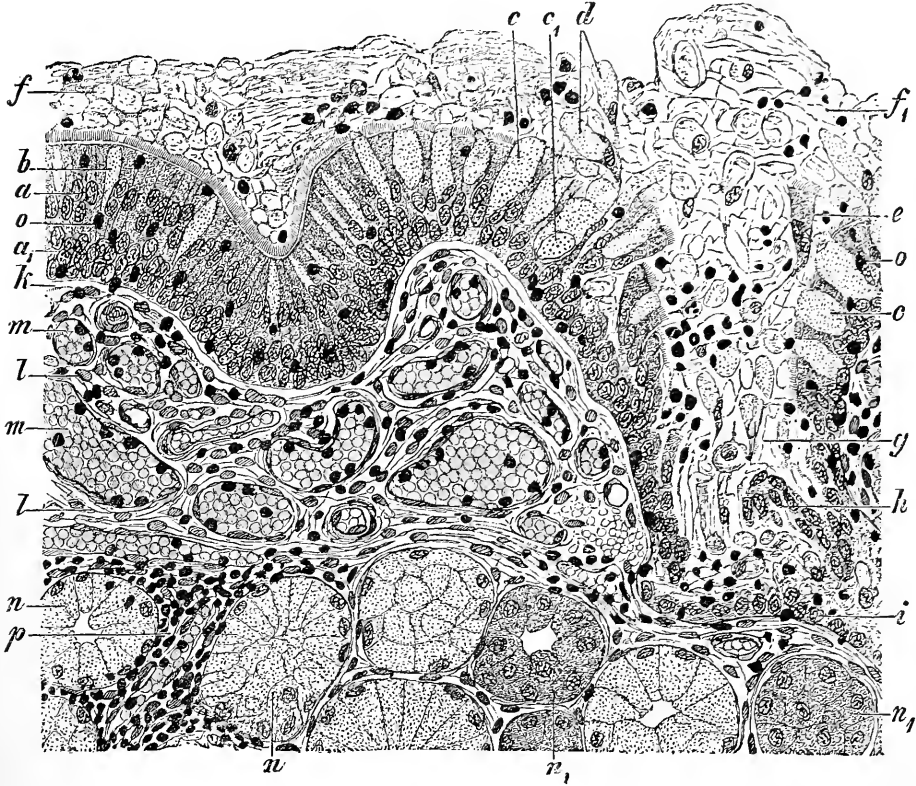


FIG. 3.—Recent catarrhal bronchitis. Section showing desquamated mucoid cells (*d*), ciliated cells (*e*), mucus-droplets (*f*), mucus-filaments and pus-corpuscles (*f*₁), in the superficial secretion, swollen basement membrane (*k*) and cellular infiltration of connective tissue of mucosa (*l*) and mucous glands (*p*). (Ziegler.)

are enlarged, indurated, and pigmented, and there is often patchy black discoloration of the lungs. Emphysema, dilatation of the right side of the heart, and chronic congestion of the liver, spleen, and kidneys are common secondary complications.

PLASTIC BRONCHITIS is a very rare affection, in which casts of the smaller bronchial tubes are expectorated during life, or are found blocking them after death. It occurs at any age, but com-

paratively seldom before fifteen or after fifty, and is twice as frequent in males as in females. Sometimes several members of the same family are affected, and sometimes groups of cases are met with, as if due to some common endemic influence. Like ordinary bronchitis, it is more prevalent during the colder months, and sometimes recurs in successive winters. Some cases have followed measles, influenza, typhoid and scarlet fever; and others have been observed to be connected with tubercle, rickets, certain skin diseases, as herpes, pemphigus, and impetigo, pregnancy, and menstruation. We have no knowledge of the real cause of the formation of the casts.

Coagula resembling the casts expectorated during life (see p. 167), or of a softer material, have been found in the tubes after death in some cases, generally loosely adherent, but sometimes quite free. The condition may be limited to a few tubes or involve a considerable number, or coagula may be absent altogether after death. The mucous membrane in different cases has been found normal, injected, or ulcerated. The bronchial wall may show the changes of chronic bronchitis. Emphysema is an almost constant feature. Tubercle, chronic heart disease, pneumonia, and pleurisy have sometimes been associated conditions.

BRONCHO-PNEUMONIA.—The part played by micro-organisms, as well as the other factors in the causation of broncho-pneumonia, has been considered in the section on general etiology. The pneumococcus, the streptococcus pyogenes, or the staphylococcus pyogenes is found in nearly every case, whether arising in connection with some specific fever or otherwise. In the so-called aspiration forms of broncho-pneumonia, where food or drink is drawn into the air-passages, germs are probably introduced along with the irritating material.

In broncho-pneumonia, inflammation extends from the small bronchioles to the alveoli, and is combined with collapse resulting from the blocking of the tubes. The lungs, both of which as a rule are affected, contain a number of areas of collapse together with more or less solid patches of irregular shape, which, as seen on section, are usually of a mottled grayish-red colour, smooth or somewhat granular-looking, slightly raised above the surface, and ill-defined at the margins. The solid patches are seldom larger than a hazel-nut, except when they result from the coalescence of a number of similar adjoining areas, when consolidation may affect a large tract, seldom, however, a whole lobe. The bronchi in relation with the solid patches contain yellowish secretion. As a rule there is little or no exudation on the pleura, even when

the consolidation extends to the surface. The patches of collapse which are present are sometimes continuous with or surround the masses of consolidation, and sometimes are quite independent. The free borders of the lungs anteriorly are usually emphysematous. The alveoli are filled with a variety of cells, chiefly large round or oval cells of epithelial origin, but small round cells, and sometimes a few red corpuscles, are also present. Fibrin filaments are, however, seldom visible, as in the lobar form of pneumonia. The bronchioles contain an exudation consisting of small round cells, and are often dilated. The epithelial cells lining the alveoli and bronchioles are swollen and proliferating, and in the case of the latter may be detached. The alveolar walls are thickened and their vessels are distended. The mucous membrane of the bronchioles is swollen, and, together with the peribronchial tissue, may be infiltrated with leucocytes. In some cases there may be little or no alveolar consolidation apparent to the naked eye, and it is to cases of this kind that the term *capillary bronchitis* has been applied.

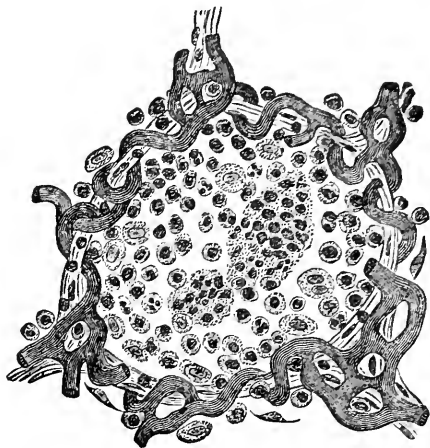


FIG. 4. — Broncho-pneumonia. Showing an alveolus filled with small round cells and large round or oval cells of epithelial origin. (Ziegler.)

Broncho-pneumonia may terminate in resolution, the cells in the alveoli undergoing fatty degeneration and disintegration, and being eliminated by expectoration or absorbed. Some cases end in suppuration and gangrene; other cases are continued as chronic pneumonia with bronchiectasis supervening later on.

It must be remembered that broncho-pneumonic processes are very often associated with the presence of pulmonary tuberculosis.

The associated lesions of broncho-pneumonia are swelling of the bronchial glands, dilatation of the right side of the heart, catarrh of the stomach and intestines, congestion of the liver and kidneys, and rarely thrombosis of the pulmonary artery or pericarditis.

ACUTE PNEUMONIA.—The specific cause of acute pneumonia is usually, if not always, the pneumococcus, as has been pointed out when discussing the general etiology. Whether the microbe reaches the lung by the air-passages or by the circulation is uncertain. In some cases the pneumococcus appears to be

accompanied or replaced by other micro-organisms, the streptococcus or Friedländer's bacillus. In the lobar or croupous form of pneumonia, inflammation starts in the alveolar capillaries, and results in the temporary consolidation of a considerable portion or the whole of one or more lobes. At first there is simply vascular engorgement of part of the lung, which thereupon speedily becomes solid from the filling of the alveoli with a fibrinous exudation, and

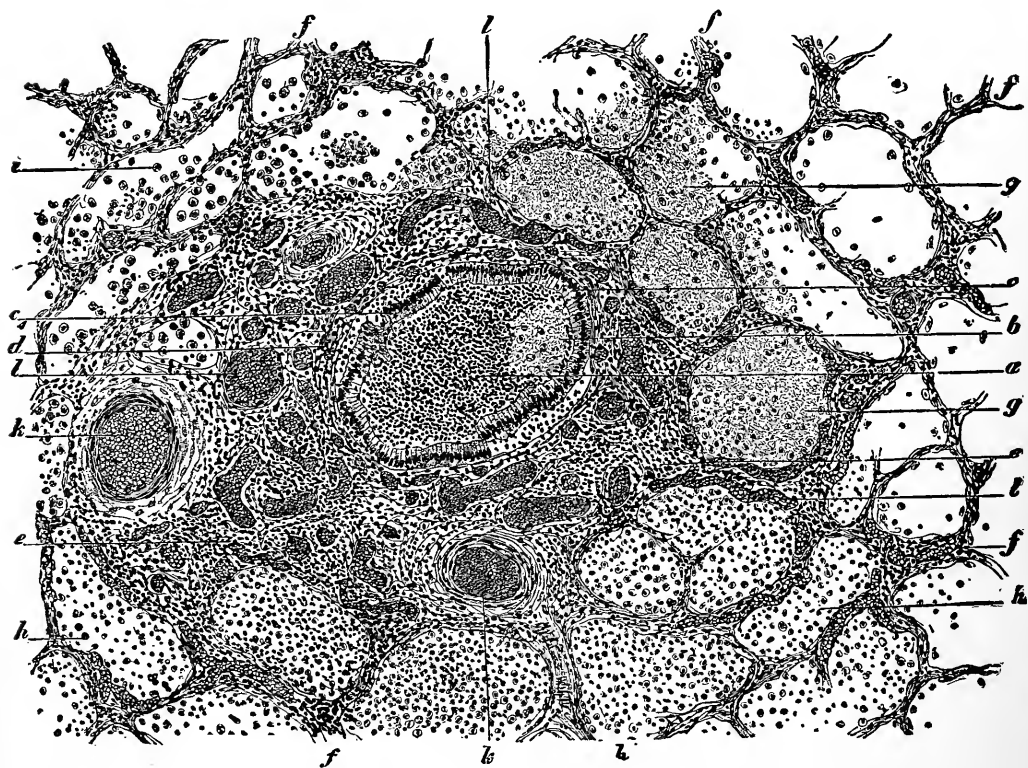


FIG. 5.—Broncho-pneumonia. Showing purulent (*a*) and mucous (*b*) secretion in bronchus, the epithelium (*cc*₁) of which is partly desquamated; bronchial wall (*d*) and peribronchial and periarterial wall (*e*) and septa (*f*) infiltrated with leucocytes; some of the alveoli contain fibrinous exudation (*g*) and others leucocytes (*h*) and (*i*). (Ziegler.)

after a time the lung returns to its original condition. There are thus three stages: congestion, consolidation—or hepatisation as it is called, from a supposed resemblance of the solid lung to liver substance,—and resolution.

In the stage of *congestion* the pulmonary capillaries are engorged; the epithelial cells lining the vesicles are swollen and granular, and the alveoli are partly filled with serous fluid containing in small proportion leucocytes, red corpuscles, and flattened epithelial cells.

The tissue is still crepitant and air-containing, although increased in density; but it has lost its elasticity and pits on pressure, while blood-stained serum exudes on section.

The stage of *consolidation* has been subdivided into a stage of "red hepatisation" and a stage of "gray hepatisation," but it is often extremely difficult to draw the line between the two. The solid portion of lung is enlarged to its fullest capacity and much increased in density. In the stage of *red hepatisation* the blood-vessels are

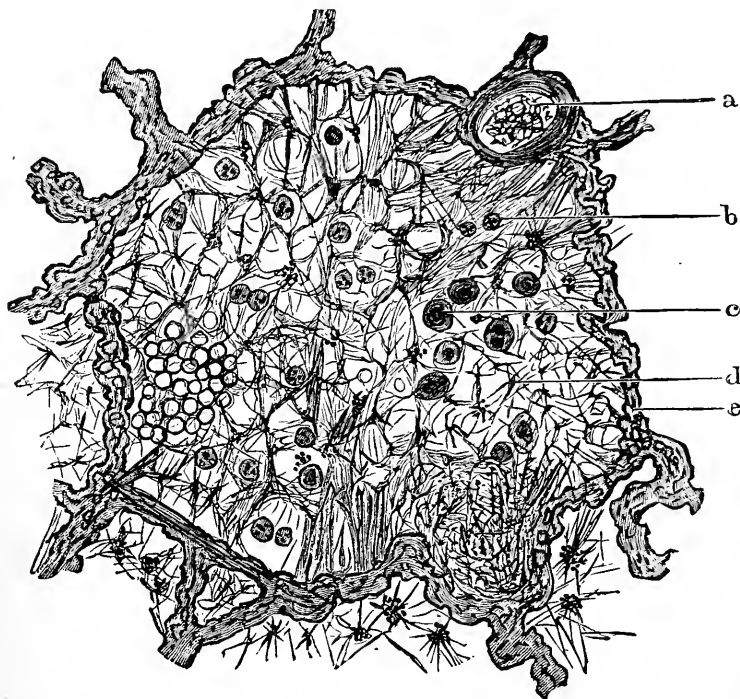


FIG. 6.—Red hepatisation. Section showing fibrinous network (*d*), leucocytes (*b*), and epithelial cells (*c*) in the exudation, swelling of alveolar wall (*e*), and engorgement of small artery (*a*).
—From *Text-Book of Pathology*. Prof. Hamilton.

still engorged, but the alveoli are filled with a fibrinous exudation containing in its meshes a large number of white cells, chiefly polynuclear leucocytes, mingled with a varying proportion of modified epithelial cells and red corpuscles, which in some cases may largely predominate. Apart from distension of the capillaries and oedema of the connective tissue, the walls of the alveoli are themselves but little affected, and the exudation may be readily separated from them. The affected tissue is solid, airless, sinks in water, and is very friable. On section it has a granular appearance, which is even more marked when the tissue is torn;

and although it looks dry, it yields on scraping a little reddish, rusty-looking fluid, which sometimes contains small plugs of fibrin. The colour is a dark reddish-brown, but is seldom uniform, there being often decidedly paler patches, the shade of which has been aptly compared to a mixture of Chinese white with red. In the stage of *gray hepatisation* the blood-vessels, instead of being engorged, are comparatively empty, the alveolar walls are infiltrated

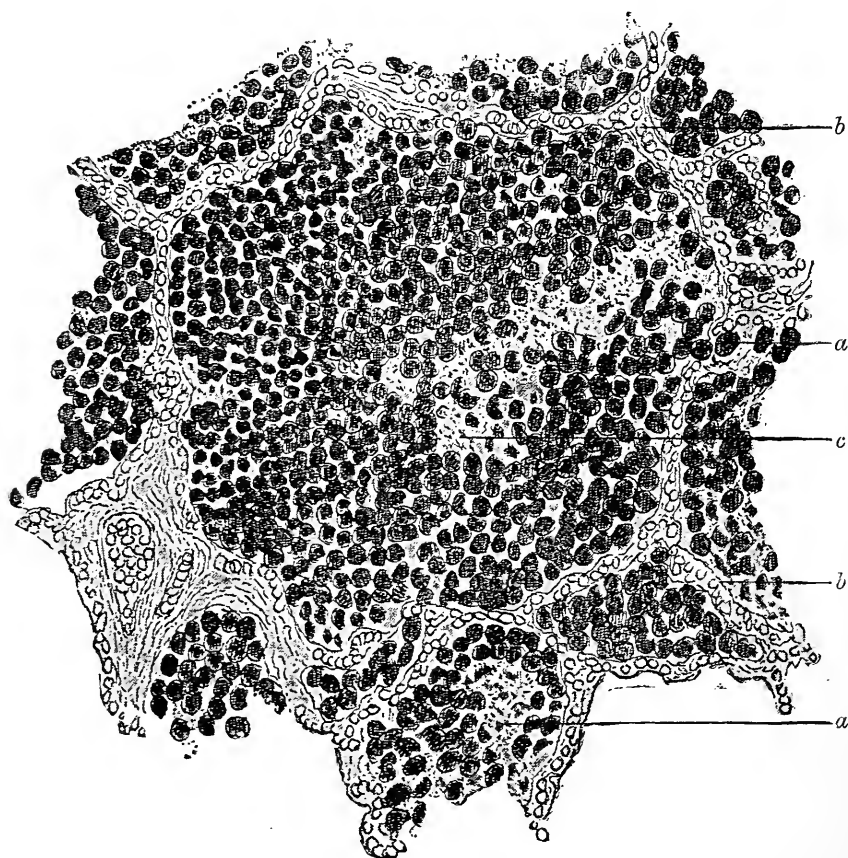


FIG. 7.—Gray hepatisation. Section showing the preponderance of leucocytes (*a*) in the exudation, the capillaries (*b*) still congested, and commencing disintegration of exudation at centre of alveolus (*c*).—From *Text-Book of Pathology*. Prof. Hamilton.

with leucocytes, and leucocytes enormously preponderate in the exudation, in which the fibrinous network and red corpuscles are no longer visible. The tissue is soft and pulpy, the section is less distinctly granular, and a puriform grayish fluid exudes on scraping. The colour is a uniform gray or grayish-yellow or green. Sometimes the two stages of hepatisation are combined, reddish patches being

so mingled with almost colourless patches as to produce a marbled appearance.

In the stage of *resolution* the lung gradually returns to its former condition. The circulation is re-established, the contents of the alveoli liquefy, and the cells undergo fatty degeneration and are removed either by absorption or expectoration.

In the first stage, the pleura covering the affected part of the lung is usually congested, and subpleural hæmorrhages may be seen beneath the surface. When the lung is consolidated the pleura over it is commonly coated with a layer of fibrinous exudation, which sometimes attains a considerable thickness, and there is generally slight effusion into the pleural cavity. Empyema is a not uncommon sequel.

Throughout the acute period the bronchial mucous membrane is injected and the lumen of the smaller and sometimes even of the larger tubes in the consolidated areas may be blocked with fibrinous casts. The bronchial glands are swollen.

The right side is more frequently affected with pneumonia than the left, and the base than the apex, which may be due to the relative extent of lung occupying these parts. Both lungs are affected in about 10 per cent of the cases.

As regards associated conditions, the non-consolidated parts of the lungs are usually found to be congested and œdematous in fatal cases. The right side of the heart is distended and filled with fibrinous coagulum, and there is sometimes recent thrombosis in the pulmonary artery. The liver and spleen as a rule are congested. Pericarditis, ulcerative endocarditis, meningitis, suppurative arthritis, and peritonitis are occasional complications or sequelæ, which, like pleurisy and empyema, arise from the invasion of the various structures by the pneumococcus. Thrombosis sometimes occurs in peripheral vessels. There may be catarrh of the intestines, and rarely ulcerative colitis or enlargement of Peyer's patches has been observed. Acute nephritis, parotitis, cellulitis, otitis, salpingitis, and keratitis are rare complications.

CHRONIC PNEUMONIA.—The term chronic pneumonia has been applied to certain conditions of lung of which fibrosis is the leading characteristic. Some of these are cases of chronic pulmonary tuberculosis, in which the fibrous change has greatly predominated. Others are cases where bronchiectasis is a marked feature. In others the induration of the lung appears to have been set up by the constant irritation of inhaled dust. Some cases in which the fibroid change has a lobar distribution have been described as

following acute pneumonia, but this course of events is very rare. Broncho-pneumonia is one of the most common causes of chronic pneumonia in children; and bronchiectasis, whether preceded or not by broncho-pneumonia, is generally associated with induration. A form has been described in which fibrosis apparently has arisen by extension from the pleura. The disease is nearly always unilateral, or, at least, much more marked on one side than the other. The organ in part or as a whole is much contracted, firm, hard, more or less deeply pigmented, traversed by dilated bronchi, and usually surrounded by much thickened and adherent pleura.

Excavations are usually of tuberculous origin. The sequelæ are usually those of bronchiectasis, which is so commonly present. Three varieties of chronic pneumonia have been described: a massive or lobar form, an insular or broncho-pneumonic form, and an irregular form, which has been termed "reticular fibrosis." In the lobar form the induration affects the whole or the greater part of a lobe, usually the lower, and to those cases following lobar pneumonia the term "subacute indurative pneumonia" has been applied. In the broncho-pneumonic form pigmented patches of fibrosis are scattered throughout the lung, the intervening tissue being either normal or emphysematous or traversed by fibrous bands. In the reticular form, which is so rare as to be a pathological curiosity, both lungs are symmetrically traversed by numerous intersecting grayish fibrous strands following the course of the interlobular septa. There is marked peribronchial thickening, with atrophy, or rarely thickening, of the mucous membrane of the bronchioles. The condition is attributed by Dr. Percy Kidd to a progressive chronic bronchitis and peribronchitis.

PNEUMOCONIOSIS.—The form of chronic pneumonia which is set up by the constant inhalation of dust has attracted much attention, and requires separate description. Indurations of variable extent replace the normal pulmonary tissue. These are sometimes massive, occupying the greater part of a lobe, or consist of nodules of varying size, from a millet-seed upwards, scattered through the lungs. The upper lobes are frequently, but neither constantly nor exclusively, affected. The indurations are almost invariably black, whatever may have been the nature of the dust inhaled, but the pigmentation is most intense in the case of miners and colliers. A brick-red coloration has been observed in persons who, in their work, have inhaled peroxide of iron. The pigmentation is due in great measure to the direct penetration of carbon or other colouring matter into the tissues, but probably to some extent depends

on altered blood pigment. The indurations consist of dense fibrous tissue growing in the walls of the vesicles and the interlobular perivascular and peribronchial tissues. The affected parts are usually much contracted, the alveoli and sometimes the bronchi also being obliterated, although the latter are more commonly dilated. Excavations have been frequently present, but it is doubtful whether they occur except from bronchiectasis, unless tuberculosis be the cause.

The cavities, which are sometimes of large size, have ragged and irregular walls, are often traversed by fibrous bands, and are surrounded by pigmented, indurated tissue. Their contents may be purulent or blood-stained, or, in the case of miners, consist of a black fluid like the matter expectorated. Sometimes caseous or calcareous masses are embedded in the indurated tissue. The pleura is commonly adherent, thickened, and pigmented. The bronchial glands are often enlarged, are generally black in colour, and may be either soft or indurated.

GANGRENE.—The causes of pulmonary gangrene are embolism, tubercle, pneumonia, broncho-pneumonia, pulmonary hæmorrhage, pressure on or perforation of the trachea or bronchi by tumours and aneurysms, trauma, and the presence of septic materials in the air passages, as in bronchiectasis and in the case of foreign bodies and immersion. It has also been observed in the course of some of the specific fevers, such as typhus, typhoid, relapsing fever, scarlet fever, measles, and smallpox, and in diabetes, scurvy, glanders, and malignant pustule. It occurs in certain forms of paralysis and of insanity, where food is apt to enter the air-passages, and in noma, cancer of the tongue, larynx, or œsophagus, and it results from extension of gangrenous processes in neighbouring parts. It has been associated with starvation, chronic renal disease, alcoholism, plumbism, mercurialism, and syphilis.

There are two forms of gangrene, diffuse and circumscribed. The diffuse form, most commonly met with as a complication of pneumonia, putrid bronchitis, or bronchiectasis, or as the result of obliteration of a branch of the pulmonary artery, may involve the greater part of a lobe. The gangrenous mass is black or greenish-black, and is sometimes firm and solid, sometimes of pulpy consistence. In the later stages it is probably always soft, and may be almost diffiuent, leaving on removal a ragged cavity. In the circumscribed form there is a definite demarcation between the affected part and the surrounding tissue. The gangrenous mass, which may occupy an area not larger than a walnut or pea, has

usually an irregular shape, and is of a blackish or greenish-brown colour, although sometimes lighter. The mass usually softens and forms a cavity with shreddy, irregular walls, containing greenish fluid or pulp, with foetid odour. The boundary of the gangrenous area is marked by a zone of deep congestion, beyond which there is œdematous lung tissue. When gangrene reaches the surface, it invariably affects the pleura, which, unprotected by any fibrinous exudation, forms a cup-shaped depression. In such cases the membrane becomes perforated, and a septic or foetid pyopneumothorax results. Abscess of the brain has complicated some cases of gangrene.

PULMONARY ABSCESS.—Among the causes of abscess of the lung are pyæmia, embolism, tubercle, acute pneumonia, the presence of foreign bodies in the bronchi, and pressure on the bronchi by tumours and aneurysms. The lung may be perforated by abscesses external to it, such as liver abscess, sub-diaphragmatic abscess, mediastinal abscess, or suppurating bronchial gland.

Pulmonary abscess is commonly multiple, as in pyæmia and in deglutition and aspiration forms of pneumonia, but may be single, as is sometimes the case when it arises in acute pneumonia or as the result of injury. It is usually of small size, between a pea and a damson, but it may be as large as an orange, or even larger. It generally consists of an irregular, ragged cavity, with purulent, sometimes necrotic, contents in the midst of softened tissue; but occasionally it is bounded by a thickened wall, which is lined with a pyogenic membrane. The embolic or metastatic abscesses resulting from pyæmia are often very numerous, and differ in appearance and situation from the other forms. They are usually superficial or subpleural, the serous membrane being covered with exudation, and they have often a wedge-like shape. When undermined, the pleura sometimes gives way, with resulting empyema or pyopneumothorax. Abscesses may also open externally or into the pericardium. Occasionally an abscess, after discharging itself through the bronchi or otherwise, cicatrises, leaving behind only a scar of fibrous tissue.

AFFECTIONS OF THE CIRCULATION

ŒDEMA OF THE LARYNX.—Œdema of the larynx, which is taken to include œdematous laryngitis, is very rare among children except as the result of injury, and most of the cases occur during the first half of adult life. The condition is sometimes primary, as when

caused (1) by local injury, such as the impaction of hard pointed foreign bodies, scalds by steam or boiling water, or the application of caustics; or (2) by the local action of some specific poison, such as that of smallpox or typhoid fever or of erysipelas or cellulitis. In the latter case, the tissues of the neck are generally involved at the same time (angina Ludovici). In rare cases œdema of the larynx has followed the administration of iodide of potassium even in small doses, or it has occurred as a manifestation of that curious transitory form of œdema known as angio-neurotic œdema.

Secondary œdema supervenes on tuberculous or syphilitic laryngitis, on malignant disease, or on any form of laryngitis in which perichondritis or ulceration plays a part. It may also arise in the course of general dropsy, as in Bright's disease or myxœdema; or it may be the result of venous congestion, caused by pressure on the superior vena cava or its branches.

The parts which become œdematous are those in which there is a distinct layer of submucous tissue, such as the epiglottis, the ventricular bands, and the aryteno-epiglottidean folds. Occasionally the œdema is infraglottic, and this form has been observed as the result of the administration of iodide of potassium.

PULMONARY ŒDEMA.—In this condition there is a transudation of serum into the interior of the alveoli and into their walls. The œdema may be general, and exist throughout the lungs, but it is more common at the bases. The term *collateral œdema* has been applied when the œdema is limited to the neighbourhood of a part affected by pneumonia, infarction, or similar conditions.

The œdematous lung is bulky, heavy, pale, and pits on pressure. It usually has a watery look, but in some cases, where there is partial consolidation, the tissue has a somewhat gelatinous appearance. It contains less air than normal, but is rarely airless. Clear or blood-stained serum mingled with air exudes freely from the cut surface on section. The occurrence of œdema is generally determined by high pulmonary tension, with disproportionate weakness of the left ventricle.

LARYNGEAL ANÆMIA AND HYPERÆMIA.—The mucous membrane of the larynx shares in the pallor of general anæmia. Local isolated anæmia of the larynx is often an early sign of laryngeal tuberculosis. Laryngeal hyperæmia is usually the result of inflammatory processes; but it may be noted that hyperæmia of the cords may sometimes be observed in the case of male adults who make much use of the voice, without any symptoms being present.

Phlebectasis laryngis is a very rare affection in which dilated

veins are to be seen on the surface of the ventricular bands, vocal cords, aryteno-epiglottidean folds, or epiglottis.

PULMONARY CONGESTION.—*Acute congestion* commonly affects both lungs, but may be partial, as in the initial stage of pneumonia, in active tuberculisation, and in so-called “acute idiopathic congestion.” Among the causes to which acute general congestion has been attributed are violent exertion, the sudden exposure of the external surface to cold, or the sudden change from cold to heat, the imbibing, while heated, of large draughts of very cold water, profound emotion, and various toxic agents. When the circulation is suddenly arrested in one part, as by embolism, the remainder of the lungs becomes intensely congested. Acute congestion is probably present in hæmoptysis from vicarious menstruation, and it is often combined with bronchitis and œdema.

“Acute idiopathic congestion,” designated by French writers as “la maladie de Woillez,” after the physician who prominently drew attention to it, is probably only a variety of acute pneumonia in which the disease terminates without passing on to complete consolidation. This is rendered likely by the clinical history, and by the fact that pneumococci are frequently found to be present in the expectoration, as well as in blood drawn from the affected part of the lung by means of a Pravaz syringe (Carrière).

Passive congestion is met with in two forms—the general or mechanical and the basic or hypostatic. General congestion is usually secondary to chronic valvular disease of the heart, and from the appearance of the lungs has been termed *brown induration*. The lungs are bulky, heavy, tough, and inelastic, and have, when freshly cut, a yellowish or reddish-brown colour. The alveolar capillaries are distended and tortuous, and there is an increase of fibrous tissue, and deposit of hæmatoidin in the epithelial cells of the alveolar walls. Some of the alveoli are blocked with enlarged pigmented epithelial cells. *Hypostatic congestion* occurs in many acute febrile diseases, and is a terminal process in many chronic illnesses as the result of failing heart and a loss of tone in the muscular coats of the pulmonary vessels, combined with the gravitation of blood to the most dependent parts. The bases of the lungs are the parts usually involved. These are dark in colour, œdematous, engorged with blood, more or less devoid of air, and smooth on section. The pleura is free from exudation, except when pneumonia has supervened, as it not infrequently does.

Hypostatic pneumonia is simply a later stage of hypostatic congestion, and is accordingly due to the same causes. The alveoli

become blocked with epithelial cells, fibrin, and serous fluid, and patches of consolidation, which later fuse together, appear in the congested tissue. A considerable portion of the lung may become solid. The solid tissue is pale red, granular on section, and very friable. Diffuse gray hepatisation or pulmonary gangrene may follow. When recovery takes place, resolution is slow.

PULMONARY APOPLEXY OR HÆMORRHAGIC INFARCTION results from hæmorrhage into the lung tissue, which is generally attributed

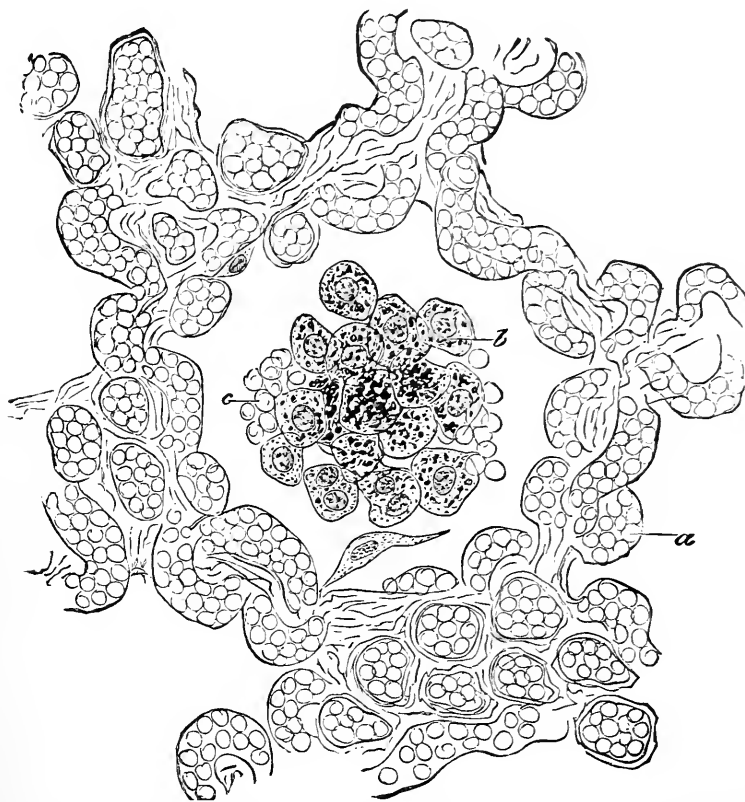


FIG. 8.—Brown induration. Showing distended capillaries (*a*), desquamated epithelium (*b*), and extravasated blood-corpuscles (*c*).—*Text-book of Pathology*. Prof. Hamilton.

to the blocking of a branch of the pulmonary artery by thrombus or embolism, and is most common in cases of mitral valvular disease, especially mitral stenosis, where long-standing chronic congestion and dilatation of the right side of the heart have existed. Professor Hamilton, however, believes that in most instances the infarction is caused not by embolism or thrombosis, but by the rupture of capillaries from over-distension, and that the wedge shape is due “not to the distribution of the terminal branches of

the pulmonary artery, but to the shape of the terminal bronchus and attached air-vesicles in which the blood is confined."

Pulmonary infarcts are pyramidal or wedge-shaped, with sharply defined borders, the base being superficial and occupying the periphery, and the apex being towards the hilus. They may be single or multiple, and vary in size from a filbert or less to a mass occupying the greater part of a lobe. When recent, they have a dark red colour, and are firm and resistant. The air-cells and finer bronchial tubes are filled with red blood-corpuscles, along with a few epithelial cells and fibrin filaments. Pneumonia may ensue, and in rare cases sloughing or gangrene has supervened. The overlying pleura is congested, dotted with small hæmorrhages, or covered with exudation; and effusion into the pleural cavity is not uncommon, especially when pneumonia has followed on. The effusion is usually simply serous, but may be hæmorrhagic or purulent, and in gangrenous cases pyopneumothorax sometimes occurs. The branch of the pulmonary artery leading to the infarct is filled with clot, which is usually non-adherent. Sometimes it is possible to recognise by its lighter colour and firmer consistence a thrombus which has been carried to the vessel from the right side of the heart or one of the systemic veins.

Recovery may take place, the effused blood being absorbed or discharged by expectoration. After a time the infarct becomes paler and of a light brown colour. A cicatrix may remain, but it is possible that no trace may be left.

PULMONARY EMBOLISM is most commonly due to the detachment of a clot from one of the systemic veins, especially the femoral, iliac, or uterine. It may also arise from the separation of part of a thrombus formed in the recesses of the columnæ carneæ of the right side of the heart. Other causes are the invasion of a vein by a mass of new growth, or by hydatid or the detachment of a phlebolith. Emboli usually lodge in the branches to the lower lobes. The smaller emboli produce the hæmorrhagic infarction, but a large embolism is often quickly fatal. In such cases it generally gives rise to œdema and sometimes to collapse. The lungs are pale and over-distended, and the right ventricle is dilated. Capillary embolisms are followed by local œdema and petechial hæmorrhages. If they come from a septic source, they produce multiple abscesses, or gangrene.

Sometimes the pulmonary capillaries throughout a certain area of the lung are blocked with fat globules (*Fat embolism*). This seriously interferes with the circulation of the blood. It has been

observed in cases of fracture of the shaft of long bones, of extensive laceration of subcutaneous adipose tissue, or of traumatic rupture of the liver. The fat globules make their way from the wounded part into the veins, by which they are carried to the lungs. When air gains admission to the veins a fatal result may quickly follow, which has been attributed to *air embolism* of the pulmonary capillaries. It is doubtful, however, whether the air is really carried to the lungs. Post-mortem examination shows great dilatation of the right ventricle, which is full of blood intimately mixed with air. The air probably remains in the right ventricle, where it is alternately compressed and allowed to expand again.

PULMONARY THROMBOSIS, besides resulting from embolism, may arise as the result of blocking of the capillaries, or from disease of the pulmonary artery. Thus it may occur in pneumonia, or more rarely when the lung is compressed by fluid or tumours, and also when the pulmonary artery is the seat of atheroma or calcification. Occasionally pulmonary thrombosis appears to precede instead of following peripheral thrombosis. Thrombosis in the pulmonary veins has occasionally been recorded, usually as the result of some preceding local lesion, such as gangrene, pleurisy, or œdema.

DILATATIONS AND OBSTRUCTIONS OF THE AIR-PASSAGES AND AIR-CELLS

BRONCHIECTASIS is rare as a primary disease, but is a condition not very uncommon as a complication of other pulmonary lesions, such as tuberculosis. Most authors speak of it as comparatively infrequent in early life, but the larger number of the cases which the present writer has seen at the Brompton Hospital and elsewhere have been in children.

Statistics would show that the male sex suffers from the disease more frequently than the female, which again has not been borne out by the present writer's experience.

The disease is invariably secondary to some disorder affecting the bronchial wall. It has accordingly been observed to follow as a sequel to bronchitis, catarrhal pneumonia, lobar pneumonia, chronic pneumonia, collapse of lung, pleurisy and pleural adhesions, obliteration of bronchi, foreign bodies in bronchi, stricture of bronchi, pressure on bronchi, and tubercle.

The following are the most important of the various theories

which have been put forward to explain the pathogenesis of the disease :—

(1) The dilatations are due to distension caused by the accumulation of mucus or other secretions in the interior of the tubes.

(2) They are hypertrophic, and are similar in origin to hypertrophies affecting other hollow organs.

(3) The dilatations are compensatory in consequence of stenosis of the larger and obliteration of the smaller bronchi.

(4) They are due to loss of elasticity and of power of muscular contraction through inflammatory changes in the bronchial wall, and are promoted by atrophy of the bronchial muscle and the dilating effects of cough acting on the weakened tissue.

(5) They are a consequence of the retraction of solidified lung and compensating enlargement of the tubes, chiefly during inspiration, to fill space occupied by retracting lung.

Of these theories the first and fourth are the most probable, and the true explanation is most likely that dilatation is the combined result of a damaged bronchial wall, accumulation of secretion, and the pressure effects of cough.

The dilatations of the bronchial tubes, which form the essential feature of bronchiectasis, may be divided into four main types :—

(1) Cylindrical or uniform, like the fingers of a glove, with somewhat bulbous endings.

(2) Fusiform, a variety of the first form, the dilatations, however, tapering at the extremities.

(3) Globular or sacculated.

(4) Moniliform or bead-like, in which the dilatations occur at intervals along the tubes.

The cylindrical and fusiform varieties are usually found affecting the larger tubules; the globular, the terminal bronchi. The dilatations may be limited to one or two tubes, but, as a rule, the disease is more or less disseminated. In some cases the whole lung is converted into a series of cavities, and resembles to some extent that of the turtle. In the smaller tubes there is the greatest proportionate dilatation. The globular dilatations are the largest, but seldom exceed the size of a hen's egg or a Tangerine orange.

The most usual seat of the disease is the base of the lungs, except in cases of tuberculous origin. In about 50 per cent of the cases both lungs are affected, although the disease is always much more extensive in one lung than in the other.

The anatomical conditions of the walls of the dilated bronchi vary. Inflammatory swelling of the mucous membrane is observed

in cases which are of acute origin, consequent on bronchitis, whether primary, or following on measles or whooping-cough. In some cases there is hypertrophy of the wall, due to fibroid changes in the sub-mucous and fibrous coats, although there is wasting of muscle, and this is common in the cylindrical dilatations of the larger tubes. But still more frequently there is thinning of the walls, which, especially in the sacculated form, may be so extreme as to make it impossible to distinguish the coats of the bronchi. The walls sometimes have a trabeculated appearance, from atrophy of the circular and longitudinal muscular fibres and their replacement by fibrous tissue. Ulceration rarely occurs, and calcification is still more uncommon. The contents of the bronchi may be similar in character to the sputum, consisting of thick fœtid muco-pus which contains ciliated epithelium mingled with leucocytes. Sometimes the tubes are empty. Sometimes the secretion is inspissated into a thick, caseous, intensely fœtid material, which may fill the tubes to distension or may be only here and there attached to the surface. The fœtor of the secretion is probably due to the influence of micro-organisms, and a special bacillus, as previously mentioned, has been found by Lumnitzer.

The changes in the bronchi in other parts of the lungs are usually those of chronic bronchitis.

The condition of the pulmonary tissue varies even more than that of the bronchial walls. In some cases it is found to be normal, in others simply compressed by the swollen bronchi, and in others emphysematous. In acute forms of dilatation, collapse is usual. Pneumonic consolidations of a lobular type are not uncommon round the dilated bronchi; but induration, often leading to marked contraction of the pulmonary tissue, as described under the heading of chronic pneumonia, is the most usual condition. When one lung is much contracted, compensatory enlargement of the other commonly takes place.

Pleural adhesions are frequently present, and in some cases attain a great thickness. They are often dense and complete when dilatation is marked, but a high degree of bronchiectasis may be found with a perfectly non-adherent pleura. Sometimes there is pleural effusion.

Gangrene of lung and perforation of the pleura are somewhat rare terminal processes. Cerebral abscess or peritonitis sometimes occurs as the result of absorption of the septic matter contained in the bronchi. Cerebral abscess from bronchiectasis is all the more serious because it is not infrequently multiple.

The associated lesions are like those of chronic bronchitis and emphysema, and consist of dilatation of the right side of the heart and chronic congestion of the liver, spleen, and kidneys. Lardaceous degeneration of the liver and kidneys has been observed.

BRONCHIOLECTASIS is a term which has been applied to what is apparently an acute dilatation of the smaller bronchioles. Most of the examples of this condition have been observed in children in cases of bronchitis and broncho-pneumonia. On account of its peculiar appearance the name "honeycomb lung" has been given to the affected organ, which on the surface shows a multitude of small air-containing vesicles, and on section has a worm-eaten appearance from the presence of innumerable small cavities, the vesicles and cavities being due to dilatation of the bronchioles, or perhaps to the breaking down of broncho-pneumonic masses. The condition usually affects an extensive area of lung, or it may be generally diffused. Microscopically it may be seen that the walls of the smaller bronchi are infiltrated with leucocytes, which pervade the peribronchial connective tissue and also invade the walls of surrounding alveoli.

EMPHYSEMA is usually the result of some disease such as whooping-cough, asthma, or bronchitis, which gives rise to cough or dyspnoea; but it may come on independently.

Occupations involving powerful expiratory efforts with a closed glottis are specially productive of emphysema. One of the earliest theories of the causation of emphysema is that it is due primarily to atrophic changes in the pulmonary tissue. There may be an inherited weakness or deficiency of the elastic tissue, or the elastic fibres may prove less resistant than usual and waste as the result of malnutrition. That inheritance has some influence appears to be borne out by the occurrence of emphysema in successive generations. The weakness may also be acquired, being induced by preceding conditions, such as bronchitis, of which atrophy of the elastic fibres is a result, and atheroma, which interferes with the nutrition of the lung; or it may result from the degenerative changes and impairment of elasticity both in the lungs and in the chest-wall consequent on advancing years.

The influence of impaired elasticity, whether inherited or acquired, must be considered as only one factor in the production of emphysema, being rather what has been called a "predisposing" and "permanence securing" cause than the actual exciting cause itself. The latter is to be found in increased intra-alveolar pressure.

Two main theories have been put forward to explain the pro-

duction of increased intra-alveolar pressure, and these at different times have been accepted as satisfactory explanations.

The older theory is that it is due to excessive inspiratory efforts, such as occur in dyspnœa from any cause. The later theory, and that which has obtained almost general acceptance, is that it is due to expiratory efforts with a closed glottis.

The increased intra-alveolar pressure may obviously occur during inspiration or expiration, or both. When the air-passages and air-cells are everywhere patent, deep inspirations act by increasing the volume of the lungs as a whole, and the lungs may become permanently over-distended if expiration be incompletely performed.

After middle age the bones and cartilages become rigid, and the thorax loses its natural elasticity, and expiration is imperfect. The inspiratory muscles usually hypertrophy on account of the greater inspiratory efforts, and the lung becomes permanently over-distended. Inspiration may tell with greater effect on some parts than others, owing to abnormalities in the chest-wall or changes in the lungs. Thus in rickets, where the sternum is thrust forward, the underlying part of the lung being unprotected becomes emphysematous, and when air is prevented from entering one part of the lung the force of inspiration falls upon the other portions.

Objections to the inspiratory theory have been made on the ground that the parts of the lungs first affected by emphysema are those in which inspiration tells with least effect. These parts are the apex, the root of the lower lobe, and the margins at the base. These are the parts which are least supported during forced expiration, and into which air is likely to be driven when prevented from escaping in the ordinary way by closure of the glottis.

The expiratory theory will explain emphysema of these portions, but it does not satisfactorily explain the production of generalised emphysema. It has been suggested that generalised emphysema arises from the inadequate support given by the intercostal spaces. As the result of the gradual alteration in the position of the ribs, different parts of the lungs are successively brought opposite to the intercostal spaces, and thus successively undergo dilatation.

Dr. Auld has observed interstitial changes in the pulmonary nerves with granular degeneration of many of the fibres, and is of opinion that emphysema may be a trophoneurosis due to the action of a poison affecting the nerve-endings in the lung. In support of this theory he mentions experiments of Brown Séquard showing that emphysema may be experimentally produced by irritation of the pneumogastric.

Several distinct types of emphysema are met with. There is an acute form which complicates acute bronchitis and other diseases where there is marked dyspnœa, and is characterised by simple dilatation of the vesicles; and there are chronic forms attended with structural changes in the alveolar walls. The condition may be local, affecting only portions of the lungs, or general, affecting the whole.

In the *acute vesicular form*, when the condition is general, the lungs completely fill the available space in the thoracic cavity, and do not collapse when removed from the body. In cases where the emphysema is local or unequally distributed, the apices of both upper and lower lobes and the anterior margins are the parts specially involved. The affected lung tissue is light, pale, and spongy, does not pit on pressure, and is compressed with difficulty. There is no structural change in the walls of the vesicles, and the over-distension would doubtless have passed off if life had been prolonged and the cause of the dyspnœa had been removed.

There are two forms of chronic emphysema: one the hypertrophous form, in which the lungs are enlarged; the other the atrophous or small-lunged form, in which they are diminished in size.

In the *chronic general hypertrophous form* the lungs are large, so that they fill the thorax, overlap the heart, and do not collapse when the cavity is opened. They are pale in colour, feel doughy, are light in the hand, pit on pressure, and are often of irregular form, with lobulated and sometimes pedunculated blebs or bladder-like protrusions from the surface. On section the lung collapses more readily than normal, and similar cavities to those on the surface are to be seen in the interior. The larger vesicular cavities vary in size from a pea to a hen's egg. At first the walls of the vesicles may be thickened, but as the disease advances atrophy occurs, the epithelial and connective tissue-cells in the alveoli undergo granular and fatty degeneration, the septa become thinned and perforated, and the capillary network in the septa is attenuated, and finally, in some places, disappears. Traces of the atrophied septa and blood-vessels are sometimes visible in the interior of some of the multilocular cavities produced by the coalescence of adjacent vesicles. In some cases the outer coat of the pulmonary vessels, the interlobular septa, and regularly recurring groups of the pulmonary alveoli are invaded by a growth of delicate new tissue of a very vascular character (Auld). Amyloid bodies are occasionally found loosely attached to the surface of the cavities or embedded in their walls.

In the *local hypertrophous form* the emphysema usually affects the apices of the upper and lower lobes, the anterior borders, and

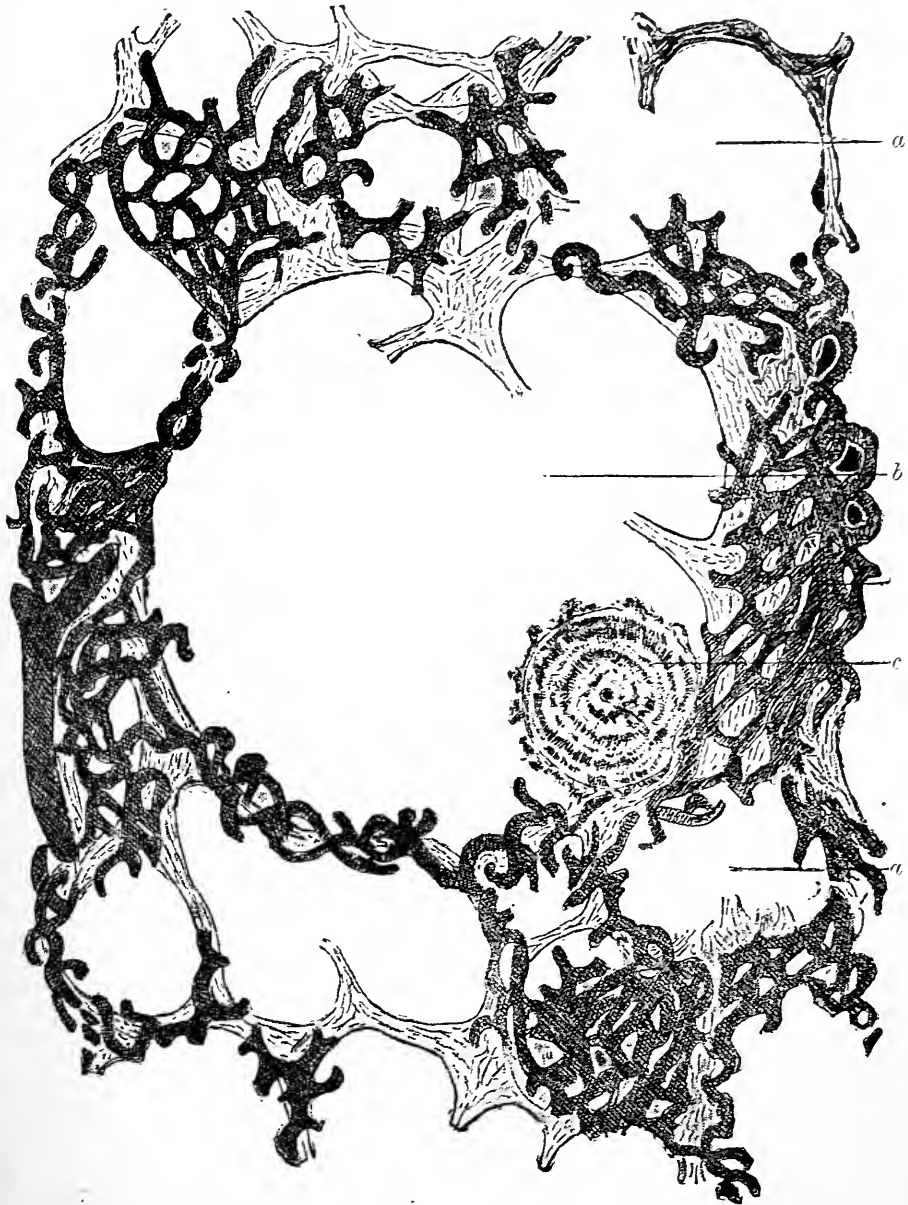


FIG. 9.—Chronic hypertrophous emphysema. Section showing dilated alveoli (a) and infundibulum (b), and amyloid body (c).—*Text-Book of Pathology*. Prof. Hamilton.

the left base. Local emphysema is not uncommonly met with surrounding localised fibrosis and obsolete tuberculous lesions. In

some cases there is dilatation not only of the vesicles but also of the infundibula and bronchioles, so that emphysema becomes combined with bronchiolectasis.

In cases where emphysema has contributed to the fatal issue it is usually associated with bronchitis, and with congestion, œdema, and sometimes collapse at the base of the lungs. There are hypertrophy and dilatation of the right side of the heart, the muscular fibre of which is often degenerated, and chronic congestion of the various other organs.

Atrophic or small-lunged emphysema.—The lungs in this condition are not enlarged, but are smaller and occupy less space than usual. They are abnormally pale, dry, and often pigmented, pit on pressure, and when the thorax is opened collapse “like bags of wet paper” (Jenner). The septa simply atrophy without any previous thickening or induration, as the result of which several vesicles fuse into one. In this form large bullæ are uncommon. The change is essentially a senile atrophy of the lung, and is usually combined with wasting of the tissues generally. The bronchi share in the atrophy, are thin and dilated, while bronchitis with puriform secretion, and collapse are frequent complications.

INTERLOBULAR OR INTERSTITIAL EMPHYSEMA is an affection quite distinct from the forms of emphysema just considered. In this condition air escapes into the connective tissue of the lungs. It may arise from the rupture of vesicles by over-distension, from wound of the lung (for example by fractured ribs), or from subcutaneous emphysema in the neck travelling down into the mediastinal connective tissue and thence to the lungs, as in tracheotomy wounds. Among the conditions in which rupture of the vesicles has occurred may be mentioned whooping-cough, severe fits of coughing, especially in laryngeal diphtheria, the violent expiratory efforts of parturition, and the dyspnœa due to pulmonary hæmorrhage. Small beads of air may be seen in the interlobular tissue, while underneath the pleura there may be blebs of considerable size. As subcutaneous emphysema may spread to the lungs by way of the mediastinal tissue, so emphysema primarily interlobular may extend to the subcutaneous tissue. Pneumothorax has occasionally resulted from the rupture of subpleural blebs. The air may be absorbed and perfect recovery take place.

LARYNGEAL STENOSIS may be caused by inflammatory swelling or œdema, the blocking of the glottis by diphtheritic membranes, new growths, syphilis, tubercle, the presence of a congenital web or

adhesions, cicatricial contraction following syphilis, injury, etc., bilateral abductor paralysis, or foreign bodies.

TRACHEAL OR BRONCHIAL STENOSIS may arise from either pressure from without or obstruction within. Tumours, enlarged glands, aneurysms, and mediastinal abscesses and hæmorrhages are the most likely causes of pressure from without. Obstruction from within may arise from swelling of the mucous membrane, polypoid growths, growths projecting into the bronchus from the lung, the impaction of foreign bodies, perforation of the bronchus by a tuberculous gland, or from cicatricial contraction due to syphilitic or tuberculous ulceration.

PULMONARY COLLAPSE.—The etiology of the congenital form of collapse termed *atelectasis* is obscure. The condition has been attributed to a considerable variety of causes, among which may be mentioned obstruction of the air-passages by mucus or meconium, pressure on the bronchi by swollen glands or hypertrophied thymus, and interference with the movements of the diaphragm by enlarged abdominal viscera, as well as pressure on the fœtal head by a narrow pelvic brim, or by the use of the forceps.

The causes of acquired collapse are:—

(1) Obstruction of the bronchi or upper air-passages due to swelling of the mucous membrane, to the presence in their interior of secretions, false membranes, new growths, or foreign bodies, to peribronchial thickening from tubercle, or to pressure from without by tumours, enlarged glands, aneurysms, etc.

(2) Pressure on the lungs by air or fluid in the pleura, by tumour or aneurysm, by an enlarged heart or pericardial effusion, or by abdominal distension.

(3) Deficient power of expansion of the lungs, from want of rigidity or softness of thoracic framework, as may be present in childhood, especially in the case of rickets; from pleural adhesions; from thoracic deformity; or from muscular weakness, the result of impaired innervation, impurity of blood, malnutrition, exhaustion from old age or disease, especially typhoid fever and dysentery, or other condition.

The frequency of collapse in children is probably largely due to the yielding character of the chest-wall and the muscular weakness, but it has been also attributed to the small calibre of the bronchioles and the ease with which they may be plugged with mucus. Collapse is especially marked where, with these conditions, there are violent expiratory efforts, as in whooping-cough.

The production of collapse by direct pressure on the lungs, as

by pleural effusion, is readily understood and requires no explanation. When there is obstruction of the bronchi, little or no air enters with each inspiration into the parts supplied by these bronchi, and after a time the residual air is absorbed. A plug of mucus may sometimes act the part of a ball-valve, preventing the entry but not interfering with the exit of air; but in many cases of collapse the obstruction of the bronchi is fluid and must hamper the entry and exit of air equally.

Atelectasis may affect a few lobules only or the whole of a lobe. It is most frequently found in the posterior parts of the lower lobes. The parts involved are of a dull purple colour, firmer than normal, airless, sinking in water, and non-crepitant. In early stages the unexpanded tissue can be inflated, and there is no reason to doubt that a lung in a state of atelectasis at birth may subsequently become completely and perfectly expanded. When the condition has continued more than a few days, inflation is difficult and requires greater effort, and after a time becomes impossible. The tissue becomes denser and firmer and has a deeply lobulated appearance. The colour at first may be darker, but it gradually becomes paler. Atrophy gradually follows, and a fibrous cicatrix may finally be the only trace left behind. Persistent patency of the foramen ovale and ductus arteriosus may be one of the consequences of atelectasis.

Acquired collapse is specially likely to affect the parts of the lungs where least expansion naturally takes place. Thus the bases, the free borders of the lower lobes, the thin anterior border of the left lung where it overlaps the heart, and the middle lobe of the right lung are the more common sites. In rickets the lung is apt to become collapsed where the chest-wall yields at the junction of the ribs and costal cartilages. Collapse is sometimes superficial; at other times it affects the deeper parts of the lungs.

When collapse is due to external pressure, as by pleural effusion, a considerable portion is usually affected, and is reduced in bulk and wrinkled on the surface. On section it has a fleshy appearance, is smooth, tough, and inelastic, and has a dark purple or bluish-gray colour, while it does not crepitate when compressed between the fingers, and sinks if placed in water. When collapse is due to bronchial obstruction, it usually occurs in scattered patches, which mostly appear on the surface as bluish-red depressed areas, surrounded by lighter-coloured, normal-looking lung tissue. On section these patches have similar appearances to those of the larger tracts of collapse due to pressure. The bronchi leading to the patches

usually contain muco-purulent secretion, and there is very often congestion and œdema in the surrounding parts. The collapsed patches are at first generally capable of being reinflated from the bronchial tubes, but as the condition persists this becomes more and more difficult. Congestion and œdema may increase so that the tissue becomes softer, more swollen, and exudes more blood-stained serum ; or the affected parts may shrink, becoming pale and indurated, resembling the lobulated whitish tissue of atelectasis. As in the congenital form, the collapsed parts may gradually expand to normal dimensions, or permanent atrophy may result, the site of the collapsed portion of lung being marked only by fibrous puckering without pigmentation. In other cases a condition similar to chronic pneumonia may supervene.

SPECIFIC INFECTIONS

SYPHILIS.—In the secondary period, catarrh of the *larynx*, shown by congestion and hyperæmia, is common, and occasionally mucous patches and superficial ulceration may be observed. No special lesions of the trachea, bronchi, or lungs occur at this stage. Tertiary lesions of the larynx are much rarer but much more serious than secondary. They consist of gummata, diffuse swelling and infiltration, perichondritis, and ulceration. The greater part of the epiglottis and arytenoid cartilages may be destroyed by caries and necrosis, or the ventricular bands, vocal cords, and aryteno-epiglottidean folds may be extensively ulcerated. Stenosis of the glottis may result from subsequent cicatricial contraction of the ulcers. Syphilis of the *trachea* is a rare affection which takes the form of a gummatous infiltration of the submucous tissue, and usually attacks the anterior surface of the tube just above the bifurcation. Softening of the gumma is followed by ulceration and perichondritis, which sometimes, as in the larynx, leads to marked stenosis due to cicatricial contraction. The trachea may become adherent to neighbouring parts, and in rare instances perforation of the œsophagus, vena cava, or aorta has occurred. Syphilis of the bronchi is even rarer than the affection of the trachea, with which, when it occurs, it is usually associated. In some cases, probably as the result of a diffuse syphilitic infiltration, both bronchi have been stenosed ; in others there has been stenosis of one bronchus, probably the result of ulceration.

Syphilis of the *lungs* is also very rare, even if we include a form

of consolidation of these organs which has been found in newly born syphilitic children. The acquired form is a late manifestation. Two kinds of lesions have been observed, gummata and fibrosis, and these may occur either separately or together. Gummata are seldom numerous, and are usually limited to one lung, of which they more frequently affect the middle portion than the apex. They may be as large as a Tangerine orange, or so small as to be scarcely visible to the naked eye, and are more often superficial than deeply seated. They consist of a firm outer part of fibrous tissue, which may contain giant cells, and a soft core consisting of yellowish material. In some cases the softened centre is discharged through a bronchus, leaving a cavity with firm walls. In other cases the gumma may dry up and its centre become calcified. The points of distinction of gummata from tubercles are their limited number, their central rather than apical position, their harder and more resistant consistence, and the absence of translucent forms.

Syphilitic fibrosis starting in the tissue surrounding the middle-sized bronchi and bronchial arteries spreads until it involves a large area of lung, the pulmonary parenchyma being gradually replaced by connective tissue of embryonic type. The apex is less frequently affected than other parts, and the affection is more commonly bilateral and diffuse, than unilateral and circumscribed. The pleura usually becomes thickened and adherent. The bronchi in some cases are stenosed or obliterated, in others dilated. The absence of evidences of tubercle and the presence of gummata or other syphilitic lesions are points of importance.

The form of consolidation affecting newly born syphilitic children, which is of little clinical importance, has been called "white pneumonia," "white hepatisation of the lungs," or "epithelioma of the lung." The lungs are usually fully distended, so that they completely fill the thoracic cavity and bear the impression of the ribs. The lung as a whole or in parts only is consolidated, the affected portions being white with a shade of yellow, dense, friable, and smooth and opaque on section. The whiteness is due to the bloodlessness, the small vessels being obliterated, but the interlobular tissue may be somewhat red. The bronchial glands are enlarged and may be partly caseous. The pleura is unaffected. The essential nature of the process is a growth of scantily fibrillated tissue in the walls of the air-vesicles, by means of which their cavities are gradually obliterated, the vessels and capillaries being completely destroyed. According to Wagner, the epithelium lining the alveoli is but little affected; but

Virchow and others have described the air-cells as being filled with epithelial cells.

It must be borne in mind that congenital syphilis is frequently associated with tuberculosis, and that many cases of apparent pulmonary syphilis are cases of tuberculosis.

TUBERCLE. — The specific cause of tuberculosis, the tubercle bacillus, has been considered at some length in the section on general etiology. It has been shown in what ways the micro-organism gains entrance to the tissues, and how other microbes may follow in its wake.

Larynx.—Laryngeal tuberculosis is usually a secondary affection, few cases occurring in which the lungs have not previously been involved. It is found to be present in a large proportion of cases of chronic pulmonary tuberculosis which terminate fatally. The proportion, which is higher in males than in females, has been variously estimated at from 24 to 48 per cent. In the majority of cases the laryngeal disease arises from infection by the secretion from diseased lung passing over or remaining in contact with the larynx. Primary cases are, however, met with, which are probably caused by the inhalation of infective dust. Tuberculous laryngitis varies greatly in the extent of the disease in different cases. Tubercles are first deposited immediately under the epithelium in the mucous membrane, which is usually markedly anæmic, and from this superficial position they gradually spread to the deeper layers. The actual tubercles themselves are often lost in a general infiltration and swelling which their presence excites. In the course of time the tubercles caseate, the overlying epithelium dies, and ulcers are left which tend to spread in depth and breadth, and may set up perichondritis, necrosis, and exfoliation of the cartilages. Any part of the larynx may be affected, but some parts suffer more frequently than others. The vocal cords, especially their posterior extremities, and the interarytenoid fold are apt to be the seat of ulceration, while swelling and infiltration invade the epiglottis and the aryteno-epiglottidean folds. The epiglottis sometimes acquires a turban-like appearance, or has a worm-eaten surface, while the aryteno-epiglottidean folds assume a characteristic pear-shaped form. In some cases a considerable portion of the epiglottis is destroyed by ulceration, but this does not happen so frequently as it does in syphilis. Occasionally tubercle leads to the formation of definite tumours. These may occur on any part of the larynx, but have most frequently been observed in the neighbourhood of the glottis. They are usually

sessile and of slow growth, and may exist without evidence of affection of the lungs.

The trachea and the bronchi are not uncommonly secondarily affected with ulceration in pulmonary tuberculosis. As a rule the ulcers are superficial and not very numerous, but sometimes the cartilages are laid bare over a considerable area, and their ragged extremities project into the interior. Sometimes destruction of the cartilages occurs, or ulceration goes on to perforation, giving rise to subcutaneous emphysema or forming a communication with the œsophagus.

PULMONARY TUBERCULOSIS.—(1) *Acute miliary pulmonary tuberculosis.*—In this form of the disease the virus has been disseminated throughout the lungs by means of the blood stream. With the naked eye it may be seen that little gray translucent nodules are scattered throughout the lungs somewhat irregularly, but present in all parts, and particularly numerous on the pleural surface. They are for the most part discrete, but may be confluent or grouped in racemose clusters. They are hard to the touch, and sometimes can be more readily felt than seen. Besides these gray miliary bodies, there are probably others with similar distribution, slightly larger and softer, opaque and yellowish at the centre or throughout, some of which may have softened, giving rise to small abscesses and cavities. Forms intermediate between the gray and yellow tubercles may also be met with. The pulmonary tissue is congested, and the nodules themselves may be surrounded by zones of hyperæmia and pigmentation. Emphysema, especially at the edges and free borders, is usually present, and patches of collapse and consolidation are to be seen, principally in the lower lobes. The solid areas may consist of ordinary red hepatisation, in which tubercles may be seen under the microscope, or of a kind of gray translucent exudation, or of an infiltration of a uniform yellow colour with discrete tubercles at the margins.

On microscopic examination the gray miliary nodules are found to consist of groups of alveoli and bronchioles filled with round granular cells in close apposition. There is diffuse thickening of the walls of the intralobular bronchioles at their terminal bifurcations, sometimes all round, sometimes at one side only. This infiltration of the bronchial wall has the ordinary structure of tubercle, for a description of which the reader is referred to Vol. I. p. 221. The elastic fibres of the alveoli can be detected, but the capillaries cannot be seen.

The main difference between the tuberculous consolidation and

that of pneumonia is that while in pneumonia the alveolar capillaries are distended and the exudation can be easily removed, in tubercle the capillaries are early obliterated and the exudation can only be separated with difficulty.

The bronchial glands are nearly always enlarged and caseous, and miliary tubercle is found in many other organs.

(2) *Acute pneumonic phthisis*.—This form of pulmonary tuberculosis is characterised by a rapidly forming caseous consolidation of the lung tissue. It is generally associated, however, with the presence of chronic lesions.

With the naked eye the lungs may be seen to contain scattered granulations such as are met with in the pulmonary form of acute miliary tuberculosis, masses of gray, red, or caseous consolidation, and cavities with caseous walls containing pus. The gray infiltration or consolidation is, in some cases, soft and somewhat granular on section, in others firm and gelatinous-looking, but is always free from any sign of injection. The red infiltration is soft, opaque, granular, and hyperæmic. The caseous tissue is yellowish or grayish-white, dry, friable, finely granular, and anæmic, neither vessels nor bronchioles being recognisable under the microscope.

The consolidation may be massive and affect the greater part of a lobe or a lung, or it may have a lobular distribution as in broncho-pneumonia. Cases differ greatly as to the number and size of cavities. The lung may be honeycombed by innumerable small cavities, or large cavities may have been formed by the fusing together of small adjacent cavities. The walls of the cavities usually show signs of active breaking down. Softening may occur immediately under the pleura and lead to its necrosis, resulting in pneumothorax.

On microscopic examination one finds changes very similar to those described under the heading of "Acute miliary pulmonary tuberculosis." In the early stage, the alveoli and bronchioles are filled with an exudation consisting of epithelial cells and small round cells lying in a meshwork of fibrin filaments. The capillaries are obliterated, and the septa and the walls of the alveoli, bronchioles, and vessels are thickened and infiltrated with small round cells. Here and there giant cells may be seen. Where the process is more advanced, caseous necrosis has caused all traces of cell structure to disappear, and the tissue has simply an amorphous or fibrillated appearance. Tubercle bacilli are generally present in large numbers in the consolidations. Clumps of them may be seen in the perivascular connective tissue, in the vessel walls, and in the peripheral alveoli.

In the non-consolidated portions of the lungs granulations of various kinds and sizes may be distributed in an irregular manner. The lungs are not uncommonly emphysematous. The emphysema may be general, or may be limited to the apices and free borders. Patches of collapse also are often met with, especially in children.

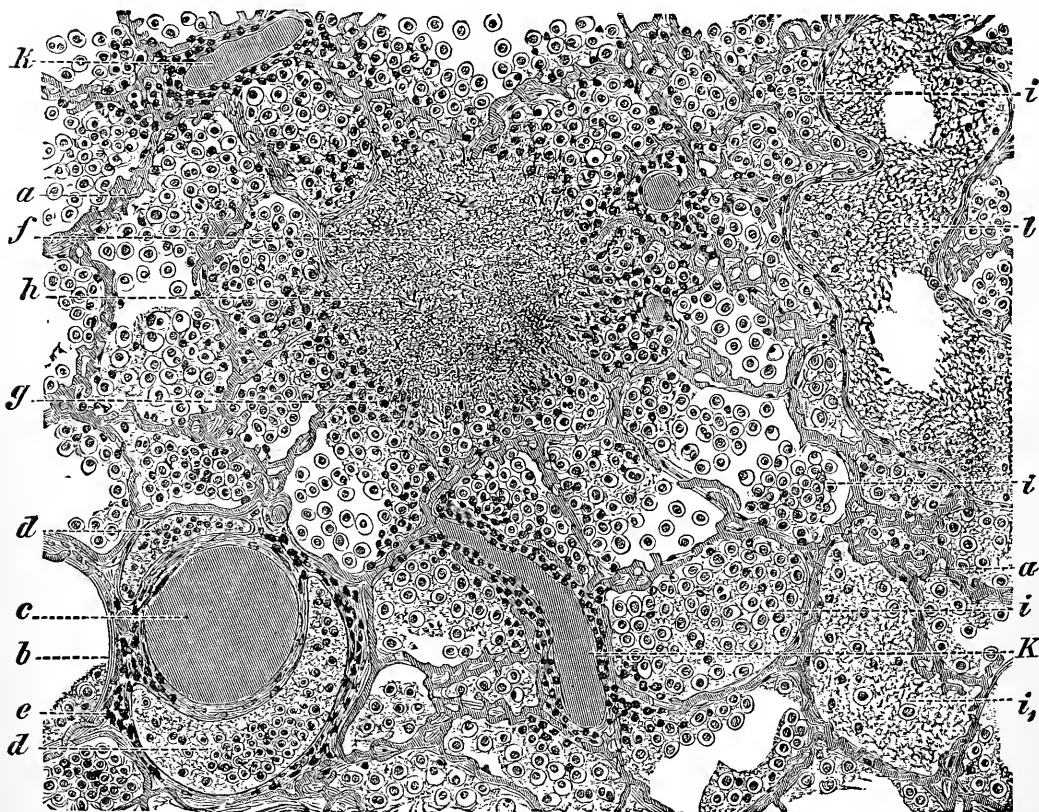


FIG. 10.—Miliary tuberculous broncho-pneumonia. Section showing caseous centre (*f*) with tubercle bacilli (*h*), and small celled infiltration at the periphery (*g*) and in neighbouring alveoli (*i*); exudation in lymphatics (*d*) and (*e*); septa (*a*), bronchiole (*b*), artery (*c*), pigment (*e*). (Ziegler.)

Tuberculous pleurisy, with or without exudation, is a common complication.

(3) *Chronic pulmonary tuberculosis*.—In this form the essential feature is fibrosis, but cavities are almost invariably present.

Chronic tuberculous disease generally begins at the apices, and there the oldest and most advanced lesions are usually to be found. The lower lobes are usually involved later, and in them the disease advances from above downwards. Both lungs are, as a rule,

affected, but one in most cases to a much greater extent than the other.

The most common situation of cavities is in the upper half of the upper lobe, but they are not at all infrequent in the lower lobes near the apices. They vary greatly in size, and in some cases a whole lobe, or even a whole lung, may be hollowed out. They may be quite superficial or more deeply situated, and rounded and independent or irregular and branching and multilocular. The cavity walls may be smooth and fibrous, or ragged and composed of softening caseous material, or they may have a trabeculated appearance from the presence of bands formed of the thickened remains of fibrous septa and obliterated vessels. These bands may run through the interior of the cavities from one side to the other. The vessels on the wall of a cavity sometimes present aneurysmal dilatations, the rupture of which is the most frequent cause of fatal hæmoptysis. Cavities may be dry or contain more or less purulent secretion. Chronic cavities are usually surrounded by indurated, gray, dense, glistening fibrous tissue, in which are frequently embedded either fibrous or caseous tubercles.

Fibrosis also occurs quite independently of excavation. At one or both apices, indurated patches of smaller or larger size may be found, which consist of strands of dense, pigmented fibrous tissue, with perhaps cretaceous nodules or dry caseous material in their interior. There are also met with consolidated patches similar to the gray infiltration of acute pneumonic phthisis, firm, smooth, glistening, and tough, containing embedded caseous nodules. Granulations of various forms, firm, dense, often deeply pigmented, and surrounded by fibrous tissue, are irregularly distributed, but with a tendency to racemose grouping.

Bronchiectasis, usually of a fusiform character, frequently occurs with chronic tuberculous lesions, and sometimes with the more acute. When the dilatation is globular, it is difficult to distinguish it from a cavity which has arisen in the ordinary way.

It must be borne in mind that in practice chronic disease is generally combined with more recent and acute processes, so that in addition to these older lesions we generally find evidences of acute miliary deposit or caseous consolidation or softening, such as have been described under the two preceding headings.

The progress of the morbid process in pulmonary tuberculosis may at any period of the course be interrupted by various complications, two of which, hæmorrhage and pneumothorax, are of a serious nature. The general pathology of pulmonary hæmorrhage

is considered at p. 75, but we may here refer to it as connected with tuberculosis. Large hæmorrhages are usually caused, as above-mentioned, by the rupture of pulmonary aneurysms. These

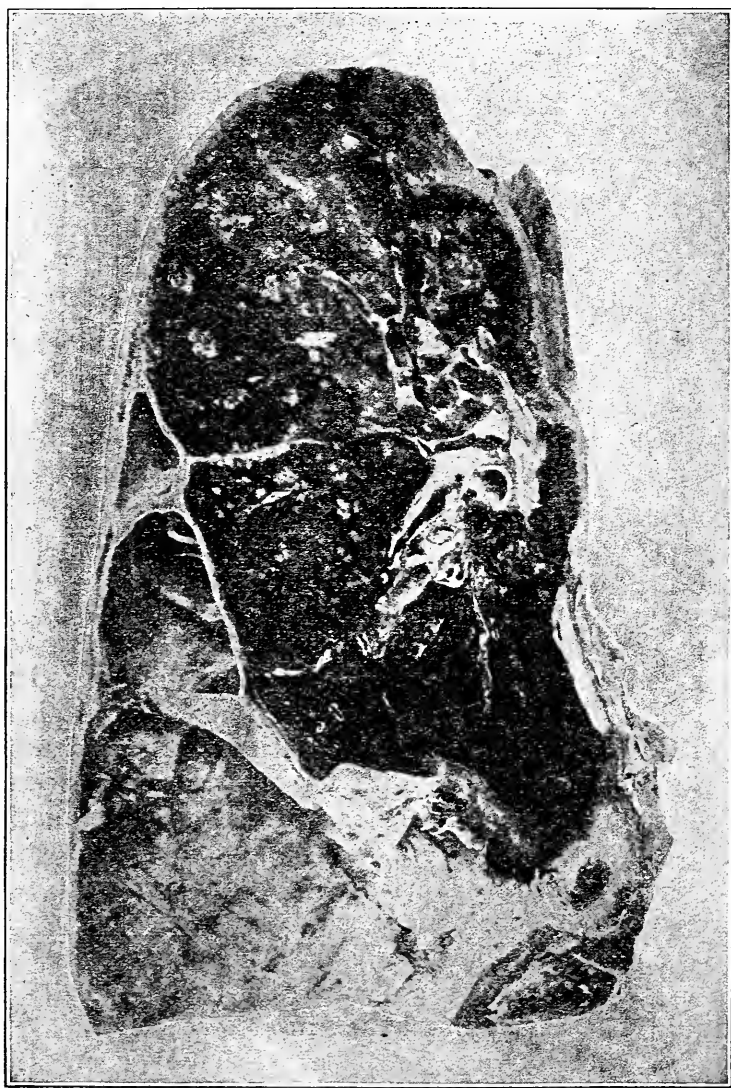


FIG. 11.—Chronic pulmonary tuberculosis with secondary pleural effusion.—From the *Catalogue of the British Congress on Tuberculosis* (1901).

aneurysms owe their origin to suppurative arteritis, which destroys the middle coat of the artery, the aneurysmal wall being formed by the expanded outer and inner coats. They appear on the vessels

lying on the walls of cavities which sometimes are but little larger than the aneurysms themselves. In some cases they are numerous and readily seen, in others one only may be discovered after a long search. They are generally about the size of a pea, but may be as large as a cherry. Sometimes a large vessel may be eroded and rupture without the formation of an aneurysm. The exact cause of hæmorrhage in the early stage of tuberculosis is somewhat uncertain. Small hæmorrhages probably arise from the rupture of capillaries in the hyperæmic areas surrounding newly formed tubercles, while the larger are probably due to the giving way of vessels in the small cavities which are usually present even at an early stage.

Pleurisy is a very common complication, and in some instances precedes by a considerable interval the signs of pulmonary tuberculosis. Serous effusion into the pleura may occur during the active stage, and not infrequently is bilateral. As the result of pleurisy, with or without effusion, the lung usually becomes adherent over a certain area, and sometimes the adhesions are universal. Local thickenings occur over contracted cavities or fibroid indurations, and may be considerable, especially at the apex. Tubercles, except on the interlobar surface, are not easily distinguished, but occasionally may appear under the form of large flat masses lying along the edges of the ribs. Were it not for the formation of adhesions over affected areas of lung, pneumothorax would be of much more common occurrence than it is. It is observed in from 8 to 10 per cent of the fatal cases. When a cavity comes to the surface, the pleura over it has its vascular supply cut off unless adhesions have already formed, and so is likely to undergo necrosis and permit of the rupture of the cavity into the pleura. For this reason pneumothorax occurs more frequently from the rupture of a small vomica due to rapid softening than from the giving way of a large slowly formed cavity.

The bronchial glands, especially those situated at the bifurcation of the trachea, are affected in a large proportion of cases. They are often enlarged. They may be caseous in whole or part, calcareous, stony, or of mortar-like consistence, or either indurated or soft, but in both cases usually pigmented. Tuberculous glands sometimes penetrate into and become embedded in the lungs, and occasionally perforate a bronchus. Tuberculous lesions in other organs are seldom absent. Those of the larynx and trachea have been already referred to. The alimentary tract becomes affected in some part of its course in a very large proportion of the cases, which is not surprising

considering that tuberculous sputum is constantly coughed up into the mouth, and in many cases is swallowed. Tuberculous disease of the tongue, which is rare (.7 per cent), most commonly occurs near the tip, and may end in destroying a considerable portion. The hard and soft palate, which are also seldom affected, may be the seat of gray granulations and ulcers. The tonsils are frequently found to be tuberculous in cases of fatal pulmonary tuberculosis. There is usually, however, no naked-eye evidence of this, but occasionally caseation and ulceration have been observed. The œsophagus is very seldom the seat of tubercle (.3 per cent), which may occur in the form of discrete nodules or ulcers. The stomach is a little more frequently affected by tuberculous ulceration than the œsophagus. The intestines are affected in some part of their course in a very large proportion of the cases which terminate fatally (70 per cent). The part most frequently involved is the ileo-cæcal region (60 per cent). In the majority of cases where the large and small intestines are affected, the frequency with which ulceration is observed is in inverse proportion to the distance from this part. In 40 per cent the vermiform appendix is affected. The appearances of tuberculous ulceration of the intestine are described elsewhere. Perforation of the intestines occurs in a little over 1 per cent of the fatal cases of pulmonary tuberculosis. Fistula in ano occurs in about 5 per cent of the cases, and probably starts with a tuberculous ulcer in the rectum near the anus. The liver and spleen seldom become secondarily attacked with tubercle, except in acute miliary tuberculosis, and then in the miliary form. Of the other organs the kidneys are most frequently affected (miliary tubercle 7 per cent). Tuberculous meningitis, pericarditis, or peritonitis are occasional complications (3 to 4 per cent).

Healed or arrested tuberculosis.—Evidences of healed tuberculosis are often found on examining the bodies of persons who have died from accident or other causes. In these cases there is usually puckering of the apex of the lung, with local thickening of the pleura. Embedded in the lung are found patches of firm pigmented fibrous tissue sometimes surrounding calcareous or caseous nodules. These arrested lesions are more commonly seen at both apices than at one only, and are often combined with evidences of old tubercle in the bronchial glands. They are observed in about 8 per cent of all fatal cases. Similar healed lesions are also found along with active or advancing disease in the case of persons dying from acute or chronic tuberculosis.

The associated lesions of chronic pulmonary tuberculosis.—

Emphysema is frequently observed surrounding patches of fibrosis, and when the upper lobe is extensively affected the base is generally emphysematous. Œdema of the lung commonly occurs in the case of acute tuberculosis, and collapse often accompanies tuberculous lesions in children.

The heart is usually small and pale in colour, and on microscopical examination the muscle may show brown atrophy or fatty degeneration. Acquired valvular disease of the heart is neither more nor less frequent than in other diseases, with the exception of mitral stenosis, which is seldom associated with phthisis; on the other hand pulmonary tuberculosis is often observed in congenital heart disease. Thrombosis of the systemic veins is not unusual as a terminal process in chronic phthisis.

The stomach and intestine are frequently affected with catarrh, and the intestine may show amyloid degeneration.

The liver is fatty in many instances (30 per cent), and less commonly shows chronic congestion or amyloid degeneration (10 per cent). Cirrhosis is present in a larger proportion of the cases than it is among adult persons dying from other causes, showing the influence of alcohol in the causation of phthisis.

The spleen may be enlarged in acute cases, and amyloid degeneration is sometimes (11 per cent) observed in chronic cases.

The kidneys may be affected with amyloid disease, chronic interstitial or parenchymatous nephritis, or congestion, but in the majority of cases they show no naked-eye changes.

ACTINOMYCOSIS AND ASPERGILLOSIS.—The pathology and morbid anatomy of these diseases have been treated of in Vol. I., pp. 243-251.

NEW GROWTHS

LARYNGEAL GROWTHS.—*Benign growths* occur most frequently in early childhood and between the ages of twenty and forty. *Malignant growths* are most common after forty, and the largest number occur in persons between forty and fifty. More males than females are affected by both forms of growth. No satisfactory cause can be assigned for their development, but in the case of malignant growths, heredity, chronic strain of the voice, and long-continued local irritation are mentioned as predisposing causes. The only *benign tumours* which are of at all common occurrence are papillomata, fibromata, and cystomata. Others, such as angioma, myxoma, ecchondroma, lipoma, adenoma, lymphoma,

and thyroid tumours, are extremely rare. The papilloma may be single or multiple, and, while usually pedunculated, is sometimes sessile. It varies in colour from a whitish-gray to a delicate pink or red, and has a granular, warty, or mulberry-like appearance. It does not infiltrate the surrounding tissue, and never grows from the interarytenoid fold. Its most common situation is the vocal cord, the anterior third of which, or the anterior commissure, is its favourite seat. It sometimes affects the ventricular bands, especially where there is multiple papillomatous degeneration, and sometimes projects from the ventricle of Morgagni, or is attached to the aryteno-epiglottidean fold or the epiglottis. The fibroma is usually single, and may be either sessile and semiglobular, or pedunculated with a short or long, slender or thick stalk. In size it varies from a coriander to a hazel-nut. It grows from one of the vocal cords, usually in its middle or anterior third. It has a smooth surface, and its colour varies from white to pinkish-red or blue. It is composed of connective tissue, and sometimes is very vascular. The cystoma arises from obstruction of the duct of one of the muciparous glands, and may be situated on the dorsal surface of the epiglottis, on one of the ventricular bands, on the ventricle, or on one of the vocal cords.

Malignant growths of the larynx may be either carcinomata or sarcomata, but the former are much the more common. According to their situation, they have been divided into extrinsic and intrinsic. The extrinsic class includes growths arising from the epiglottis, the aryteno-epiglottidean folds, the arytenoids, the interarytenoid fold, and the posterior surface of the cricoid, and of these the latter is the most frequent situation. The intrinsic class includes growths arising from the vocal cords, the ventricular bands, the ventricles of Morgagni, and the subglottic region. The majority of growths are intrinsic, and a large number of these start in the vocal cords. Most of the cancerous growths are epitheliomata.

GROWTHS OF THE TRACHEA are very rare. A few cases of benign growth, papilloma and fibroma, and of malignant growth, carcinoma and sarcoma, have been recorded. The trachea may be affected by extension of growth from neighbouring parts, such as the œsophagus, mediastinum, or thyroid gland.

GROWTHS OF THE LUNGS are seldom primary, and more commonly arise by extension from the mediastinum or œsophagus, or as secondary deposits from primary tumours elsewhere. Of cases of carcinoma affecting various parts of the body, secondary tumours in the lungs have been found post-mortem in about 14 per cent.

Primary carcinoma may originate in the epithelium or mucous glands of the bronchi, or in the alveolar wall. Sarcoma, of which the spindle-celled variety is the most common, is nearly always secondary, the seat of the primary disease being usually in the bones. Primary growths may appear either as definite tumours, or, as is more frequent, in the form of infiltrations. There may be one or several large tumours, hard, whitish, solid, lardaceous-looking in some cases, soft and fleshy in others. The infiltrations spread along the bronchi and vessels by means of the lymphatic spaces, or run in the course of the interlobular septa and gradually replace and destroy the proper pulmonary tissue. Sometimes there is great thickening of the pleura, or the growths appear beneath the pleura as flat, waxy-looking masses, or, penetrating more deeply into the lung, have a cupped appearance. The lung is increased in weight, and may be enlarged; but contraction is not uncommon in the infiltrating form, especially when the pleura is much thickened. Secondary tumours are usually multiple and rounded, but are sometimes small, numerous, and widely disseminated, in which case they closely resemble tubercle. Abscesses and cavities sometimes arise from the softening of cancerous masses.

In cases of primary cancer of the lung it is rare to find secondary growths in other organs.

HYDATID OF THE LUNG is a very rare affection in this country and in America, although less uncommon in Australia and Iceland. Statistics show that in this country 5.9 per cent of all cases of hydatids are pulmonary or pleural (8 out of 136 cases, Cobbold); in America 6.2 per cent (15 out of 241 cases, Lyon); in France 10.6 per cent (40 out of 376 cases, Davaine); and in Australia 14.8 per cent (271 out of 1897 cases, Davies Thomas). Hydatid of the lung is due, as is hydatid disease in other parts of the body, with which it is not uncommonly combined, to the introduction into the body by ingestion or inhalation of the ova of the *Tænia echinococcus* of the dog. It is probable that inhalation is the usual method of infection in the case of primary pulmonary hydatids. The pulmonary hydatid, which may attain a considerable size, is usually single, but more than one may exist, or both lungs may be affected. The cyst itself presents the same characters in the lung as elsewhere, but the adventitious fibrous capsule, the line of defence set up by the body against the parasite, is not usually so dense as in other organs, or may be wanting altogether. The hydatid tumour as it grows displaces, compresses, and erodes the lung tissue, and thus open bronchi may

be in direct communication with the surface of the tumour and hæmorrhage may occur. The cyst may rupture into a bronchus or into the pleura or pericardium. By its pressure it may cause pneumonia or gangrene. Inflammatory changes in the fibrous capsule may lead to suppuration or fœtid decomposition.

GENERAL SYMPTOMATOLOGY

PYREXIA

Pyrexia is usually present in the acute affections of the respiratory organs, such as acute bronchitis, bronchopneumonia, pneumonia, pleurisy, empyema, pulmonary abscess or gangrene, and acute pulmonary tuberculosis, and occurs not infrequently at some stage or other of the chronic disorders, such as bronchiectasis, chronic pulmonary tuberculosis, new growths, and pulmonary hydatids. The subject of pyrexia in relation to disease in general has been considered in Vol. I. (pp. 50-58). The pyrexia of respiratory disorders is due to the introduction of some poison into the blood. Either the blood has been infected from the local disorder as in the case of chronic phthisis, or the local disorder is itself the result of a general systemic infection as in the case of pneumonia. The characters of the pyrexia will be considered in each case in association with the particular disease in which it arises. Here there may be appropriately considered certain modifications of pyrexia which are common to all morbid affections of the respiratory organs. In young children temperatures tend to run high, and a degree of pyrexia which in later life would be of grave significance may occur in them, in pneumonia or bronchopneumonia for example, without special danger. In persons of all ages, debilitated by previous disease, cachexia, or alcoholism, during an attack of acute illness such as pneumonia, temperatures tend to be lower than in those previously healthy and robust. In old persons, also, during the course of an acute respiratory disease, temperatures are usually not quite so high as in persons affected in earlier life, but this is generally due to previously enfeebled health. Rigors, which are of common occurrence at the onset of acute disorders, like pneumonia, in previously healthy adults, may be absent or less marked in old people. The reason for this, however, is also probably not so much age as previous debility, for in a hale and vigorous old person the onset of pneumonia may be marked by a violent and prolonged rigor.

In old persons the surface of the body tends to be colder than in younger subjects, although, in the case of old people who are healthy and vigorous, the temperature of the internal organs is usually up to the normal standard. For this reason axillary temperatures are not altogether to be relied on, and observations of the rectal temperature are more trustworthy. In some cases of pulmonary tuberculosis more accurate information as to the progress of the disease is to be obtained when temperatures are systematically taken in the rectum.

COUGH

Cough is one of the most constant symptoms of disease of the respiratory system. It is characterised by the sudden forcing of air by expiratory effort through the partially closed glottis, with or without expectoration. Closure of the glottis is preceded by a preliminary deep-drawn inspiration, and then followed by a sudden forced expiration or series of expirations, in which the ribs are rapidly drawn downwards and inwards, the lungs being thus forcibly compressed, the air driven through the trachea and glottis, and the contents of the lower air-passages expelled. Cough may be reflex or voluntary, and in either case is generally preceded by a feeling of tickling or discomfort in the larynx or trachea. The most common cause of cough is irritation of the mucous membrane of the pharynx, larynx, trachea, or bronchi; but the most sensitive of these parts is the larynx, especially the interarytenoid fold. Irritation is caused by swelling, congestion, or inflammation, or the presence of abnormal secretion, such as serum, mucus, pus, or blood, or of foreign matter, such as dust. The term "reflex cough" is sometimes used in a special sense to denote the cough which is set up by distant irritation. Among the causes of such reflex cough may be mentioned a chill to the surface of the body or a sudden draught of cold air. Reflex irritation of the nasal mucous membrane may set up coughing instead of sneezing. It is well known that coughing may arise from irritation of the external meatus of the ear by wax or foreign body, and until the cause of irritation is removed may prove troublesome and persistent. The afferent path in this case is probably along the auriculo-temporal branch of the fifth nerve. Irritation of the costal pleura has been shown experimentally, and is well known clinically, to give rise to cough, a symptom of pleurisy which may cause no little suffering. Irritation from various parts of the alimentary tract may excite cough, and of this the presence of a carious tooth or stump in the mouth or of intestinal worms in the intestines may be mentioned as examples. A "stomach cough" is often spoken of by the laity, and its existence is more believed in by them than by the profession. In phthisical subjects we often observe how cough is directly excited by the presence of food in the stomach, and not uncommonly in such cases ends in vomiting. Uterine irritation is a cause of reflex cough. Thus it is well known what a troublesome symptom cough may be during pregnancy. It arises without any signs or any other symptoms of pulmonary disease, is often spasmodic, and from its paroxysmal character may raise a suspicion of whooping-cough. Cough may also be excited by stimulation of the vagus in the neck, or of the superior laryngeal nerve by tumour, abscess, or aneurysm, or of the nerve centres in the medulla

by toxic agents. The cough centre appears to be situated a little above the inspiratory centre.

The pharynx should always be examined as a matter of routine in cases of cough. Chronic pharyngitis is a common cause of irritative, hacking cough with little or no secretion. Although undue importance has been attached to the uvula as a cause of cough, there is no doubt that occasionally, when elongated and relaxed, it may so irritate the pharynx as to give rise to severe and persistent cough, which is aggravated in the recumbent position.

In its relation to the different diseases of the respiratory system cough will be considered again under the description of the respective maladies of which it is a symptom.

In different cases the frequency of cough varies widely, and it may come on at longer or shorter intervals. In some affections it is worse on waking in the morning, or in the evening when the patient is trying to get to sleep. That it is troublesome in the morning is due to the necessity of getting rid of the accumulated secretions of the night, as in cases of phthisis, bronchitis, and bronchiectasis. That it is worse at night is due partly to the recumbent position, and partly to the increased excitability of the cough centre at the period between waking and sleeping.

The actual attacks of coughing may consist of one or two short, slight, but sudden expiratory efforts occurring at intervals, or of a series of short, more severe coughs following one another; or there may be a constant and rapid succession of forcible expirations interrupted from time to time by deep inspirations. Cough is not infrequently beneficial, and in the first form is often the result of a natural effort to clear the air-passages, which it succeeds in doing, or it may be simply due to a nervous habit. In all diseases of the respiratory organs the second variety is the most common. In phthisis, the first form often alternates with the second. The third form (*tussis convulsiva*) occurs principally in whooping-cough. When cough from any cause is prolonged and severe, it is apt to be followed by vomiting.

Cough may be dry or moist, that is, without or with expectoration, if we exclude the cases where the sputum is swallowed. It may be dry either because there is no secretion, as at the beginning of bronchial catarrh or whooping-cough, and when it is a nervous or distant reflex phenomenon; or because, although there is some secretion, it cannot be dislodged, as in cases where there is some mechanical obstruction to its discharge, or where the secretion is viscid and tenacious and limited to the smaller tubes. It is obvious that the more deeply situated the part from which the secretion comes, the more troublesome and difficult will be the process of removing it.

The noises attending coughing vary both in loudness and character. As a rule the inspiratory part of a cough is noiseless. The exceptions

are the inspiratory whoop of whooping-cough, the whistling sound which occurs in laryngismus stridulus, and the wheezy, harsh inspiration which may be observed when the larynx is obstructed by membrane. When the lungs can be fully inflated, and the vocal cords are normal, the expiratory efforts are more or less noisy. The character of the noise is modified by the degree of tension of the cords, and by local changes in the larynx. Very noisy coughs are principally met with in cases of laryngeal and tracheal catarrh, and in hysterical or nervous patients. A peculiar loud cough sometimes affects adolescent subjects of either sex, and has been described as the "barking cough of puberty" (see page 152). The vocal cords may be prevented from meeting by swelling or thickening, and the cough may be noiseless. A peculiar noisy, brassy quality of the cough is characteristic of cases where there is pressure on the trachea by aneurysm or tumour.

During and after a paroxysm of cough there is swelling of the jugular and facial veins, and the patient may get quite blue in the face. Sometimes hæmorrhages occur from the nose or beneath the conjunctiva, and rarely the membrana tympani has been ruptured. Sometimes the patient is seized with giddiness, and in a severe attack may fall down insensible.

A paroxysm of cough temporarily raises the blood pressure, and impedes the entrance of blood into the heart. It lessens the amount of oxygen and increases the amount of carbonic acid in the blood. Through these results, cough, if long continued, will lead to permanent damage to the tissues. Persistent cough is one of the most potent causes of emphysema, and is then likely to lead to dilatation of the right side of the heart and chronic congestion of the various organs.

SPUTUM OR EXPECTORATION

The mucous membrane of the air-passages is normally protected from the irritation of dust or other matter by a thin layer of mucus secreted by the mucous glands. In health a small quantity of mucus may be expectorated, and this, in the case of dwellers in towns, is often black from the presence of carbon particles. In disease of the respiratory tract, the secretion of the mucous membrane is greatly increased, and may be mingled with leucocytes, blood-cells, fragments of pulmonary tissue, etc. A study of the expectoration often throws light on the processes going on in the lungs and bronchi, and is an aid to prognosis as well as to diagnosis, serving to indicate in some measure the progress of the case.

Quantity.—In disease the total quantity of the expectoration in the twenty-four hours varies from less than a teaspoonful to twenty ounces or even two or three pints. It is largest in cases of bronchiectasis, chronic bronchitis, pulmonary cavities, and empyemata communicating with the lungs.

COMPOSITION.—Chemically the sputum is composed of water, mucin, and nuclein, which are usually in large proportion, together with serum-albumin, peptone (when pus-cells are abundant), inorganic matter, fat and occasionally volatile fatty acids, glycogen, and ferments.

The principal inorganic constituents are chlorides of sodium and magnesium, sulphates of sodium and calcium, phosphates and carbonates of sodium, calcium, and magnesium, and occasionally silicates. The reaction of the sputum is always alkaline.

Consistence.—In consistence it varies widely, being in some cases thin and watery, in others thick but diffuent, or extremely viscous. Viscosity is proportional to the amount of mucus present, which depends on the degree of irritation of the mucous membrane. Very fluid, watery sputa become uniformly blended together. Very tough sputa are irregularly globular, and when expectorated into water appear as discrete masses of a flattened, rounded shape, and are then called “nummular.” Although nummular sputa often proceed from cavities, they are met with in other cases, such as chronic bronchitis.

VARIETIES.—Clinically sputum is divided into mucous, muco-purulent, purulent, serous, and sanguineous varieties, all of which are in some degree aerated.

Mucous sputum is clear, glairy, tough, and sticky, and consists principally of mucin with more or less water. It occurs chiefly in the early stage of bronchial catarrh.

Muco-purulent sputum is yellow or greenish-yellow and of different degrees of consistence, arising from the mixture of mucus and pus in various proportions. This is the most common form of sputum, and is found in a great variety of conditions.

Purulent sputum is usually diffuent, yellowish or greenish, and is met with in cases of suppurating cavities, bronchiectasis, and some forms of bronchitis.

Serous sputum is thin, very watery, usually frothy, and not uncommonly blood-stained. It occurs in cases of pulmonary œdema.

Sanguineous sputum.—The sputum may consist of pure blood, as described under the heading of hæmoptysis, or blood may be more or less intimately mingled with the sputum, or may appear in it in the form of streaks or dots. As the result of mixture with blood or blood-colouring matter, sputum acquires characteristic tints. When there is an intimate mixture, then the blood must have remained for some little time in the lungs, and the more tenacious the mucus the longer is the time necessary. When blood appears in the form of streaks, it usually comes from the air-passages. When blood remains in the air-passages mingled with the sputum, it gradually changes colour, passing through the following shades: reddish-brown, yellowish-red, yellowish-saffron, yellowish-green, green, from the higher oxidation of the hæmoglobin. The reddish-brown colour is illustrated in the *rusty sputum* of pneumonia

at the height of the disease, while the yellowish or greenish colours are observed later.

Prune-juice sputum is the name given to sputum of a thin consistence, frothy, and of a dark reddish-brown colour, which is observed in grave cases of pneumonia and pulmonary œdema, and in cases of pulmonary growth. In this form of sputum, serum-albumin is abundant. In some cases of pulmonary growth the sputum resembles "red-currant jelly."

The sputum may be bile-stained, but only when there is well-marked jaundice or a communication between the liver and the lung.

The sputum in *hepato-pulmonary abscess* has characteristic appearances, being of a dirty brick-red or chocolate-brown colour, and somewhat thick and viscid.

Black sputum occurs in coal-miners and others who inhale black dust or soot particles, and hence its common occurrence.

The ODOUR of sputum is generally not very marked or characteristic. It is variously described as faint, mawkish, or mouldy. In certain diseases, however, it becomes horribly offensive, as in bronchiectasis, gangrene, and occasionally also in pulmonary abscess. The fœtor is usually noticeable also in the breath, especially after the patient coughs. Care must be taken to distinguish fœtor due to lung disease from that proceeding from morbid conditions of mouth, nose, or stomach.

MICROSCOPICAL CONSTITUENTS OF THE SPUTUM.—1. *Leucocytes* are always present, but vary greatly in number. They form the chief constituent in purulent expectoration. Eosinophile cells are found in asthma and in some cases of bronchitis and phthisis.

2. *Red corpuscles* occur in considerable number and show the ordinary appearance under the microscope, in cases where the sputum is blood-stained.

3. *Epithelium*.—Squamous pavement epithelium from the mouth is not uncommonly present. Columnar epithelium is rare, and the ciliated variety is still more so. It may come either from the nasal passages or the trachea. Oval cells with single nuclei, containing finely granular protoplasm and sometimes granules of altered blood pigment, carbon, or iron dust, have been regarded as alveolar epithelium, but their origin from the epithelium is doubtful. They have been considered to be significant of alveolar catarrh.

4. *Elastic fibres*.—The presence of these is of great importance as pointing to breaking down of lung tissue. They occur singly or in bundles, but it is only when they have an alveolar arrangement that they are of diagnostic importance. They are slightly curved and show a double contour. To find them the sputum should be boiled with a ten per cent solution of caustic potash, and the mixture, well diluted with water, left to stand in a conical glass for twenty-four hours. The sedi-

ment, which will contain elastic fibres if these are present, is then examined under the microscope.

5. *Fibrinous casts*.—These are of a whitish-yellow colour, and have the characteristic branching appearance of the tubes in which they are formed. They occur in pneumonia and plastic bronchitis. In pneumonia they are usually small and few, and point, if numerous, to a severe form of the disease. In plastic bronchitis (see p. 167) they are longer and more minutely branched.

6. *Curschmann's spirals*.—These spiral bodies, which are chiefly met with in asthma, will be found described (at page 178) in the account given of that disease.

7. *Connective tissue, etc.*—Shreds of connective tissue are sometimes found when ulcerative processes are in progress in the lungs. In ulceration of the larynx or trachea, fragments of cartilage may be expectorated. Sometimes corpora amylacea have been observed, but their significance is unknown.

8. *Crystals*.—*Charcot-Leyden crystals*.—These are colourless, pointed octahedra met with chiefly in asthma (see p. 178). They have also been found in acute and plastic bronchitis.

Hæmatoidin crystals.—These are ruby-red, rhombic prisms due to blood retained in the air-passages, and are occasionally observed after hæmoptysis from any cause, or in cases of pulmonary abscess or liver abscess burrowing through the lungs.

Cholesterin crystals, fatty crystals, tyrosin crystals, oxalate of lime, and triple phosphate are sometimes found, but have little pathological or clinical importance. Fatty crystals, which are observed mostly in bronchiectasis and pulmonary gangrene, take the form of long, slender, colourless, lance-shaped needles, single or in tufts, straight or curved, and may be arranged so as to resemble elastic tissue. From this they can be readily distinguished by the fact that they are dissolved by chloroform or ether, but are insoluble in water and acids.

9. *Parasites*.—In cases of hydatid disease the characteristic hooklets may be discovered in the sputum. The ova of the distoma pulmonale or Ringeri (see Vol. II. p. 7) are found in the endemic hæmoptysis prevalent in certain parts of Japan, Corea, and Formosa. The thrush fungus, *oidium albicans*, may be present when there are aphthæ in the mouth. *Leptothrix* has been observed in cases of putrid bronchitis.

The most important of all the constituents of the sputum are those micro-organisms which are the actual specific causes of the diseases in which they are met with. The discovery in the sputum of the tubercle bacillus, the pneumococcus, the streptococcus pyogenes, or the fungus of actinomycosis or aspergillosis, is of the highest value. It is unnecessary here to repeat the description given elsewhere of these micro-organisms, but we may refer to the methods of staining the sputum so as to reveal their presence.

In staining for tubercle bacilli, it is first desirable to select a likely portion of the sputum. A small purulent fragment should be picked up with a needle, as bacilli will more likely be present in this than in the mucus. In cases of hæmoptysis, one of the small dark-red clots to be observed as the attack is subsiding should be chosen. If the sputum be spread on a clear shallow vessel placed on a black background, a suitable fragment will be more readily obtained. The fragment is then deposited on the centre of a clean cover slip, another clean slip placed on the top, and the two are firmly pressed together so as to distribute the sputum evenly over their surfaces, after which they are separated by a sliding movement. The slips are then passed through the flame of a Bunsen burner three times, which fixes the albuminous coating. Of the many methods of staining the following is one of the simplest and best. The staining fluid (Ziehl's carbol fuchsin) consists of 10 c.c. of a solution of one part of fuchsin in ten parts of absolute alcohol added to 100 c.c. of a 5 per cent watery solution of phenol. The staining fluid is heated in a watch-glass placed on a sand-bath until steam rises. The cover glasses are then floated film downwards on the surface of the staining fluid for about four minutes. They are then removed by means of forceps, and placed in a 25 per cent solution of nitric acid, in which they are left until all colour disappears. The slips are next washed in tap water, and are counter-stained in a saturated solution of methylene blue, after which they are washed again in tap water and allowed to dry, and may then be mounted in Canada balsam. The tubercle bacilli are stained red, while the various cells are coloured blue. (See Vol. I. Plate II. Fig. 10.)

To stain for pneumococci a rusty-coloured portion is selected, and a thin film is dried and fixed on a cover slip, as already described. It is then stained by Ziehl's method and decolorised in warm water. The cocci are darkly stained, while the capsule shows a fainter shade of red. (See Vol. I. Plate I. Fig. 3.)

In cases of suspected actinomycosis the sputum should be spread out on a dark plate; one of the small pale-yellow globular granules should be then picked out with a needle and placed on a clean slide with a little water. A clean cover slip is then put over it, and the specimen slightly flattened by pressing with the point of a needle. On examination with the microscope the characteristic ray fungus may be seen. If preferred, the specimen may be previously stained by Gram's method.

HÆMOPTYSIS

Hæmoptysis, or blood spitting or expectoration, includes the discharge of blood from any part of the air-passages from the larynx to the alveoli. Hæmorrhage from the larynx is uncommon. In acute laryngitis the scanty mucous expectoration may be streaked with blood.

Larger hæmorrhages may occur in cases where there is ulceration, whether tuberculous, syphilitic, or malignant, or in altered blood states. Most of the recorded cases of hæmorrhage in laryngitis have been among women, especially pregnant or recently confined women, or persons with degenerated vessels. Large hæmorrhages from the trachea and bronchi, except in cases of aneurysm pressing on and causing atrophy of the walls, are still more uncommon, but may occur in cases of ulceration from any cause. In cases of bronchitis and tracheitis the expectoration may be stained with blood. In much the largest proportion of cases hæmoptysis is due to hæmorrhage from the lungs, of which by far the most common cause is pulmonary tuberculosis. A large hæmorrhage usually arises from the rupture of an aneurysm of one of the small branches of the pulmonary artery situated in a pulmonary cavity. Vessels, however, may rupture without the previous formation of aneurysms. Smaller hæmorrhages in the lungs may result from diapedesis or from the rupture of capillaries when there is active or passive hyperæmia. Active hyperæmia is the cause of the rusty or prune-juice sputum of pneumonia, and of the capillary hæmorrhages which are common in the earlier or active stages of pulmonary tuberculosis when miliary infiltration is in progress. Passive hyperæmia is the chief agent in the production of the hæmorrhages which occur in chronic cardiac disease, especially mitral stenosis, although pulmonary embolism or thrombosis is usually the direct cause of the larger hæmorrhages of heart disease. Pulmonary hæmorrhage may also result from the ulcerative processes associated with the presence in the lungs of new growths, hydatids, actinomycosis, aspergillosis, or distoma Ringeri, or caused by bronchiectasis, gangrene, abscess of the lung, pulmonohepatic abscess, or perforating empyema. Among other causes may be mentioned injuries of the thorax, the rupture of an aortic aneurysm, blood conditions such as hæmophilia, purpura, and scurvy, vascular degenerations of old age, and emphysema and vicarious menstruation.

Hæmorrhage from the lungs is more common among males than among females. Children and old people are relatively less subject to it. In some persons there appears to be an inherited weakness of the vascular tissue and a natural tendency to hæmorrhage. Hot weather predisposes to it, and it is found that phthisical patients suffer more from hæmoptysis in summer than in winter. Diminished atmospheric pressure has been given as a cause of hæmoptysis, but it is doubtful whether its occurrence under such conditions has not been accidental.

Blood when expectorated in any quantity is generally bright red, fluid and frothy, in contrast with blood vomited from the stomach, which is usually dark and clotted. When bleeding takes place from the lungs, the blood is brought up almost immediately, as contrasted with hæmatemesis, where the patient usually turns pale, feels faint and

squeamish, and then after an interval vomits up the blood. There is a greater liability to syncope in hæmatemesis than in hæmoptysis. Bleeding from the lungs usually subsides very gradually, and the patient, after ceasing to cough up pure blood, expectorates blood-stained sputa for some time. Hæmorrhage from the stomach is rarely repeated, and is generally large in amount. It may, however, occur again and again, but usually at considerable intervals, and when it stops it does so abruptly.

Hæmorrhage from the pharynx may give rise to a suspicion of hæmoptysis. It is characteristic, however, that it occurs only early in the morning, that it is small in amount, and is not associated with any constitutional disturbance. In hysterical women a form of blood-spitting is sometimes met with which has been termed "pseudo-hæmoptysis" or "spurious hæmoptysis." In cases of this kind the patient spits a thin, watery, blood-stained fluid, which is simply saliva mingled with a small quantity of blood. The source of the bleeding may be the gums or the back of the mouth. Care should be taken not to mistake epistaxis for hæmoptysis.

As has been mentioned, pulmonary tuberculosis is by far the most common cause of hæmoptysis, and evidences of tubercle should be carefully looked for, repeated physical examinations and bacteriological researches being made in doubtful cases. In tuberculosis the smaller hæmorrhages, usually capillary, occur during the active stages of the disease. Hæmorrhage is often coincident with fresh advance, to which it may directly contribute by sowing new infection in the lungs. In such cases systematic observations of the temperature are very important, as activity of the disease is accompanied by pyrexia. The larger hæmorrhages which come almost invariably from diseased vessels in the walls of cavities often occur during periods of quiescence of the disease.

DYSPNŒA

Dyspnœa or difficulty of breathing is a symptom which occurs in many other diseases besides those directly connected with the respiratory system, but we shall here consider it in its special relation with the latter. In dyspnœa the breathing is altered, either in rate, or in depth, or in rhythm, or in all three. Dyspnœa may be subjective or objective. It is subjective when the person feels short of breath; it is objective when we observe that there is alteration in the rate or depth of the breathing. There may be objective dyspnœa without the patient being himself conscious of any difficulty of breathing.

The average rate of respirations per minute in the healthy male adult is from 14 to 18; in women slightly more. The normal ratio of the respiration rate to the pulse rate is about 1 to 4. In some diseases this ratio is markedly altered. It is important to remember that

respiration is normally more rapid in children. Under one year the average rate is 44, and between one and five, 24 to 32. In old persons there is an increase in the number of respirations and a diminished amount of carbon dioxide exhaled. Respiration is slightly more rapid in the standing than in the sitting, and in the sitting than in the recumbent posture, and is least rapid during quiet sleep. It is increased by bodily exertion, markedly so by running, and thus a person who is free from dyspnœa while at rest may at once feel short of breath on exertion. This is the case in some of the forms of lung disease, such as chronic phthisis. Idiosyncrasy seems to play a certain part in the production of dyspnœa. In some persons otherwise in good health and free from pulmonary disease, the least unusual exertion may habitually bring on a sense of dyspnœa. It has been suggested that in these cases "either the excess breathing power is always low or there is an undue sensitiveness of the respiratory reflexes" (Gairdner). In other individuals very extensive pulmonary disease may be present with comparatively little breathlessness.

The following are among the chief causes of dyspnœa :—

(1) The breathing of air which, on account of deficiency in oxygen, excess of carbon dioxide, or the presence of organic or other impurities, is unfit for respiration.

(2) Obstruction to the air-passages, the nose, larynx, trachea, or bronchi, whereby the free entrance of air to the lungs is interfered with.

(3) Diminished extent of the respiratory surface as the result of pulmonary disease, as for example in pneumonia where the pulmonary alveoli are blocked with exudation, in pleuritic effusion or pneumothorax where the alveoli are compressed, or in emphysema where the air-cells have lost their elasticity and power of expansion.

(4) Interference with the free movements of respiration either by defective innervation, as in paralysis of the diaphragm or intercostals, or by changes in the thorax, or by upward pressure on the diaphragm, from ascites, enlarged liver or spleen, abdominal tumours, the pregnant uterus, or meteorism.

(5) The presence of any obstacle to the free circulation of blood through the lungs, leading to its insufficient oxygenation, arising either from pulmonary disease or imperfect cardiac action (cardiac dyspnœa).

(6) Alterations in the quality or quantity of the blood whereby its power of taking up oxygen is lessened, as in all conditions in which either the number of the red corpuscles is reduced, or there is a diminution in the amount of hæmoglobin they contain, such as anæmia in its various forms, leucocythæmia, etc., and after loss of blood by hæmorrhage.

(7) The action of toxic agents circulating in the blood on the respiratory centre in the medulla, as in fevers, renal disease (renal

dyspnœa), and various other maladies, such as diabetes and acute yellow atrophy of the liver.

(8) Involvement of the respiratory centre or its connections in diseases of the nervous system such as apoplexy, or perversion of its action, as in so-called hysterical dyspnœa.

(9) The influence of pain in the thorax or abdomen, on account of the presence of which the respirations are abnormally superficial and hurried.

(10) Elevation of temperature brings about increased rapidity of breathing. The rapid breathing of fever is partly due to this cause. Animals artificially heated suffer from rapidity of respiration, or, as it has been called, "heat dyspnœa."

The kind of dyspnœa which arises from diminished amount of oxygen in the air is illustrated by the curious malady known as mountain-sickness or *mal des montagnes*. It has been found experimentally that when the oxygen in the air is reduced to 10 per cent dyspnœa arises. This attenuation is reached at a pressure of 330 mm. Hg., corresponding to a height of between 16,000 and 17,000 feet. It is found in practice that at this height the breathing becomes gasping and hurried, and there is a strong craving for air, which is inhaled with open mouth in gulps (Vol. I. p. 19).

Although an increase in the amount of carbon dioxide in the air has been considered a potent cause for dyspnœa, it is very doubtful whether it has any such action provided the amount of oxygen remains of normal amount (see p. 24).

Dyspnœa may be chiefly *inspiratory* or chiefly *expiratory*, or a combination of both. It takes the inspiratory form when there is obstruction of the air-passages, and this form attains the highest degree when the obstruction is in the larynx. It takes the expiratory form where there is some impediment to the free escape of air from the lungs, or where the lungs are over-distended and have lost their elasticity, permanently or temporarily, as in emphysema and asthma. In the inspiratory form the auxiliary muscles of inspiration are specially brought into action; in the expiratory form the abdominal muscles are principally used. In inspiratory difficulty the scaleni and sterno-mastoids are first called into play, and the latter muscles may be seen standing out in the neck. The pectorals, which raise the ribs when the head and shoulders are fixed, and the scapular muscles are used in more severe cases. When dyspnœa is severe the patient assumes the sitting posture (*orthopnœa*), as in this way the diaphragm is relieved of the pressure of the abdominal organs, and the inspiratory and expiratory muscles can be employed to greater advantage. In some cases, in order to obtain leverage for the auxiliary muscles, he fixes the upper part of the chest by leaning over or holding on to a table or other support.

In slight degrees of expiratory dyspnœa, expiration is more slowly

and gradually performed than normally, and there is no apparent pause between the end of expiration and the beginning of inspiration (Chart 2). In a more severe form, the lungs are never completely emptied, inspiration starts at a higher level, the auxiliary muscles of inspiration are brought into play, so that the chest is over-expanded, and expiration is slowly and laboriously performed (Chart 3). In a still more severe form, the chest is fully distended at the commencement of inspiration, the inspiratory movements are entirely due to the auxiliary muscles, and while expiration is still prolonged the respiration is quicker than normal (Chart 4). In the most severe form of all, the chest is over-distended throughout, and the movements are extremely slight (Chart 5).

When dyspnœa arises from obstruction in the air-passages to the free entry of air, the breathing is more or less noisy. When the seat of obstruction is in the nose, the breathing has a sniffing or bubbling character; when due to paralysis or relaxation of the soft palate, it is snoring or stertorous; when in the glottis, it is stridulous; when in the trachea, rattling or growling; and when in the bronchi, wheezing.

The most severe forms of inspiratory dyspnœa, it has been pointed out, are due to laryngeal obstruction. It is sometimes difficult to determine whether the obstruction is in the larynx or trachea, and we may mention the contention of Gerhardt that when dyspnœa is due to obstruction in the larynx it is attended by rapid and extensive movements of the larynx, which do not occur when the obstruction is in the lower air-passages, and that in the former case the head is thrown backwards, while in the latter it is stretched forwards.

In the form of dyspnœa due to paralysis of the diaphragm (*phrenic dyspnœa*) the breathing is easy while the patient remains at rest, but on the slightest exertion or excitement it becomes hurried and embarrassed. The voice and cough are feeble, and the acts of coughing, sneezing, and defæcation are attended with difficulty. While the movements of the thorax are increased, there is reversed action of the wall of the abdomen, which is drawn in in inspiration and pushed forwards during expiration.

Various toxic substances will produce dyspnœa through their action on the respiratory centre. The dyspnœa which attends exertion is attributed to the presence in the blood of some poisonous substance, the product of tissue metabolism.

The form of dyspnœa which precedes or accompanies diabetic coma, and is observed in some other diseases, such as acute yellow atrophy of the liver, is characteristic, and has been aptly termed "air hunger." The rate of respiration is little if at all affected, but both inspiration and expiration are unusually deep and prolonged, and the breathing not uncommonly has a panting or sighing character, while a moan or groan may accompany expiration.

Respiration may be retarded instead of being accelerated in cases of stenosis of the larynx or trachea, and in some forms of brain disease.

Chart 1. *Respiration curve normal in respect of both rhythm and respiration levels.*

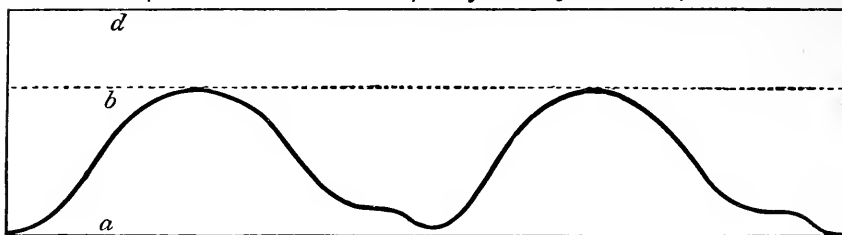


Chart 2. *Respiration curve in slight emphysema. Expiratory portion of curve more gradual in descent.*

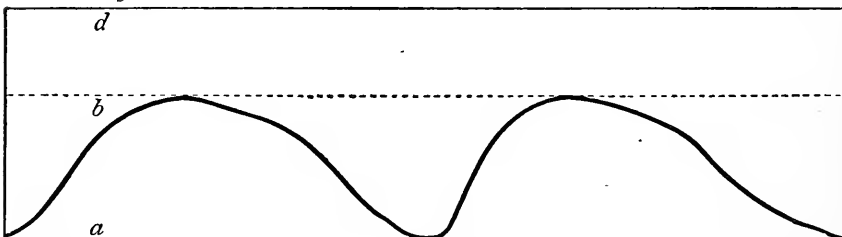


Chart 3. *Respiration curve in more severe emphysema during an attack of asthma of moderate severity. Similar to 2, but respiration at higher level.*

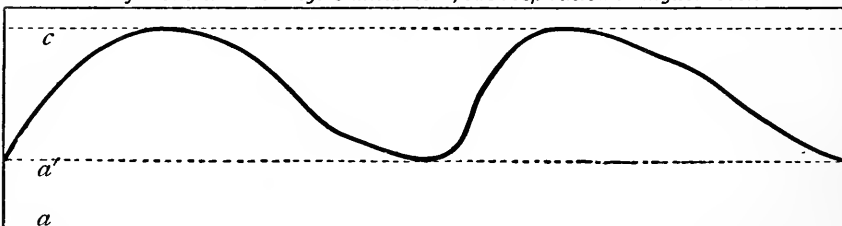


Chart 4. *Respiration curve during severe attack of asthma with emphysema. Respiration quickened and level still higher than in 3.*

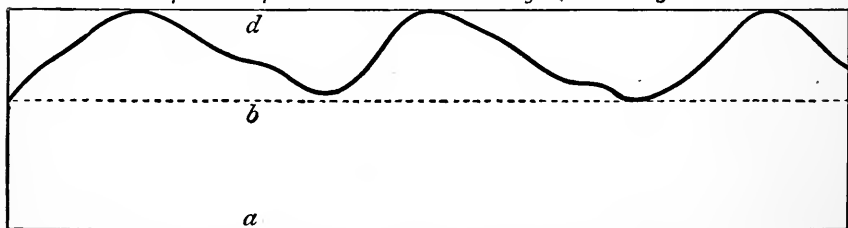
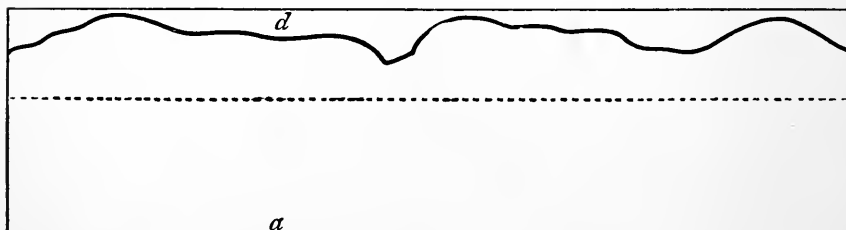


Chart 5. *Respiration curve during an intense attack of asthma. Extreme chest distension. Range of movement very limited. Expiratory movement almost nil.*



a. Level of normal expiration. *a'* Higher level of expiration in asthma.
b. Level of normal inspiration. *c.* Level of extraordinary inspiration.
d. Level of extreme inspiration.

Under certain conditions a peculiar "tidal" or "periodic" form of dyspnœa comes on, which is known as *Cheyne Stokes breathing*. This kind of breathing arises in the course of very diverse maladies, including diseases of the brain, lungs, and heart, and various forms of toxæmia; and, although most frequently the forerunner of death, it sometimes passes away and the patient recovers. It usually continues for not longer than forty-eight hours, but cases have been recorded where this type of breathing has been maintained for months or years. It is characterised by the occurrence of regularly recurring pauses, during which the respirations are completely suspended for from five to forty seconds. After a pause the breathing starts again with short, shallow, feeble inspirations, which gradually become deeper and quicker until they have a dyspnœic character, when again they gradually become slower and more superficial, until they cease altogether and another pause occurs. The dyspnœic period usually occupies from thirty to forty-five seconds, during which time the respirations may number about thirty. The cause of Cheyne Stokes breathing is obscure. It has been attributed to exhaustion of the respiratory centre, when the respiratory movements, no longer under guiding control, have a tendency common to all vital functions to become periodic. In a form of breathing allied to the Cheyne Stokes type the respirations, instead of beginning gradually after the pause, start at once fully and deeply, and then gradually subside.

In hysterical subjects respirations may be extremely rapid—forty, sixty, or even eighty to the minute—over a long period of time without any of the usual causes of dyspnœa being present. In these cases the breathing usually falls to a normal rate during sleep.

Clinically it is important to make the distinction between dyspnœa of gradual and of acute onset. Where the cause of dyspnœa is of slow and gradual onset, as in chronic pulmonary diseases, then dyspnœa itself develops slowly. Where the cause is due to some rapid change in the respiratory organs, then the dyspnœa comes on suddenly. Instances of dyspnœa of sudden onset are pneumothorax, where a whole lung is suddenly placed *hors de combat* from collapse; and pulmonary embolism, where the circulation through a large section of the lungs is suddenly interrupted. Dyspnœa comes on rapidly in acute pulmonary diseases, but seldom with absolute suddenness. The dyspnœa of asthma is of rapid development, and so is that caused by the intermittent pressure of a tumour or aneurysm on the trachea.

DISORDERS OF CIRCULATION

The presence of disease in the lungs almost invariably produces secondary effects on the heart and circulation. The obliteration of large numbers of capillaries in the lung as the result of emphysema, fibrosis,

excavation, or prolonged compression, leads to hypertrophy and dilatation of the right side of the heart, and the same consequences of backward pressure, such as œdema and the congestion of the various organs, which follow primary cardiac disease. Thus it may be sometimes difficult to distinguish a case of chronic bronchitis with secondary dilatation of the right side of the heart from a case of bronchitis secondary to mitral valvular disease.

Changes in the rate and force of the pulse are of frequent occurrence. A slow pulse is sometimes observed in asthma. A rapid pulse in pulmonary disease is generally associated with pyrexia ; but in the case of phthisis it is not uncommonly observed where the temperature does not rise above normal, and, when persistent, must be considered an unfavourable sign. In other cases the heart's action may be only rapid on exertion, as in emphysema. Attacks of palpitation are not uncommon in emphysema and in chronic phthisis. Extreme degrees of rapidity often accompanied with irregular action may be observed in bronchopneumonia in very young children. Irregularity and intermittence of the pulse are common in pulmonary diseases in old people. Inequalities of pulse on the two sides may be present in the case of thoracic tumours.

Since the intrathoracic pressure falls during inspiration and rises during expiration, the pulse tends to be weaker during inspiration and stronger during expiration. When the inspiratory negative pressure becomes abnormally low, as it may do in the case of stenosis of the larger air-passages, then during inspiration the pulse may become very feeble or altogether imperceptible. This phenomenon, which is called the "pulsus paradoxus," is also sometimes observed in adherent pericardium or indurative mediastinitis.

Congestion of the veins, especially those in the neck, is observed in chronic bronchitis and emphysema, partly because the contractile power of the right ventricle is diminished, partly as the result of laboured and prolonged expiration which increases the intrathoracic pressure, and partly in the case of emphysema on account of the atrophy and obliteration of the pulmonary capillaries. Coughing renders the veins still more prominent. In the case of tumours which press on the intrathoracic veins, distension of the veins in the upper part of the body may be extremely marked.

As the result of chronic congestion of the veins, the face, especially the nose, ears, and lips, may become swollen and livid, the eyes prominent, and the conjunctivæ congested. The superficial veins on the chest, the mammary, and superior epigastric, may become varicose and tortuous.

CYANOSIS

Cyanosis is a frequent result of dyspnœa, and is due to a deficiency of oxygen and excess of carbon dioxide in the arterial blood, which excess

in some cases is as great as or greater than that in venous blood. In cyanosis there is an increased number of blood-corpuscles, which has been ascribed to diminished metabolism and lessened wear and tear of the blood-cells. The causes of cyanosis are the same as those of dyspnœa, but there may be a high degree of cyanosis without marked subjective dyspnœa being present. In respiratory disorders cyanosis is greatest when dyspnœa is of sudden or rapid onset, and when the patient is of the plethoric type. Thus pneumothorax, which comes on suddenly, produces a much greater degree of dyspnœa and cyanosis than a pleural effusion of gradual onset, although the latter may produce compression of lung to an equal degree. This may be accounted for by the compensation effected, when the cause acts slowly, in the gradual expansion of the healthy lung. In chronic phthisis, where the changes take place slowly, cyanosis is seldom marked. Children are on the whole less affected with cyanosis than adults. Plethoric subjects are more likely to become cyanotic than the anæmic, for their vessels are well filled, and oxygenation of the blood takes place more slowly. Cyanosis may be extreme in the plethoric subjects of emphysema complicated with bronchitis.

CLUBBING OF THE FINGERS AND TOES

Clubbing of the fingers and toes is a deformity which, when it occurs, is commonly associated with certain diseases of the respiratory organs. It consists as a rule of a rounded bulbous enlargement and thickening of the pulp of the terminal phalanges, while the nail is incurved towards the palm of the hand. The end of the finger or toe has thus a distant resemblance to the rounded head of a club. Sometimes with the enlargement there is flattening, and the shape is like that of a serpent's head. The skin over the root of the nail is shiny and tense. Usually all the fingers and toes are similarly affected, but sometimes, especially in advanced cases, it is more marked in the thumb and index finger. The exact nature of the change, which is entirely limited to the soft tissues, the finger-pads, and the beds of the nails, is still uncertain. Fibrous thickening of the rete mucosum has been described. Although clubbing is met with principally in pulmonary diseases, it occurs in a very marked form in congenital heart disease, and has been observed in lardaceous disease, in cirrhosis of the liver, and even in cases where no disease was apparently present. Its cause is unknown, but it seems probable that it depends on the absorption of some poison into the blood, and not on circulatory disturbance such as venous congestion. It occurs to a marked degree in bronchiectasis, and in a moderate degree it is fairly common in chronic phthisis. It is sometimes met with in pulmonary abscess and empyema, and may supervene very rapidly. It may disappear completely with the removal of the cause, as on the cure of

an empyema. When due to general causes, clubbing is bilateral and symmetrical. Unilateral clubbing has been recorded in a case of subclavian aneurysm occurring on the same side as the tumour.

Under conditions similar to those which give rise to clubbing of the fingers, certain changes in the bones and joints have occasionally been observed. To these changes the name "hypertrophic pulmonary osteoarthropathy" has been given, and under this heading a description of them is given in Vol. II. p. 268.

PAIN

Pain occurs in various diseases of the respiratory system, and is sometimes the symptom from which the patient first suffers, and that for which he seeks the aid of the physician. Pain in the chest, however, not infrequently arises quite independently of any intrathoracic disease. It may be due to rheumatism of the muscles or fasciæ, to intercostal neuralgia or neuritis, to flatulence or disorder of the stomach, to abscess or other disease of the liver, or to disease of the vertebræ, ribs or sternum. Following Dr. Head, whose researches on the character, distribution, and significance of pain of the thoracic as of other organs have greatly added to our knowledge, we may distinguish two distinct varieties of pain, "local" and "referred" or "reflected."

Local pain is situated directly over the focus of disease in certain cases, being limited to one area and not distributed in a band. It is especially marked in the intercostal spaces, and when uncomplicated is unaccompanied by superficial tenderness, such as is elicited by pinching up the skin, but is associated with tenderness to deep pressure or percussion.

Local pain is met with in the neighbourhood of tuberculous or pneumonic consolidations and pulmonary infarctions, and generally is associated with the presence of dry pleurisy. It is of a stabbing or catching character, in abeyance while the side is still, and aggravated by taking a deep breath. As already mentioned, deep tenderness is present, and at some point of the area of its distribution friction will usually be found to be present. The deep tenderness is probably due to pressure on the inflamed pleura. Sometimes in pleurisy the intercostal trunks are involved, and pain may then be felt over their terminations in the abdomen or iliac fossa. In this case the pain is not felt over the back like referred pain, and although superficial tenderness may exist it is only present on the distal side of the patch of pleurisy.

Referred pain, on the other hand, is not necessarily situated over the affected part. It runs round or through the body, and has two foci of greater intensity, one behind and the other in front. When severe or at all persistent, it is accompanied by superficial tenderness over certain definite spots or areas.

Referred pain is due to the "distension of a sensitive organ from within, or by the exertion upon it of some tearing or rending strain from without." No referred pain can result unless the end organs or some of them within the diseased area are intact. Any disease which arrests the movement or destroys the tissues, including the nerve-endings of the affected part, will be without referred pain. Hence acute pneumonia and rapidly spreading tuberculous caseation and chronic phthisis with extensive fibrosis or excavation are, as a rule, without referred pain. Referred pains are of the type usually spoken of as intercostal neuralgia, myalgia, or intercostal rheumatism, and are described as stabbing, catching, aching, or tight. The pain is not, as a rule, increased by deep breathing, but by anything which throws more work on the diseased lung, such as running up a flight of stairs. The pain does not follow the course of one intercostal nerve, but may affect parts of the areas of several.

The superficial tenderness may be recognised and mapped out by picking up the superficial structures between the finger and thumb, which manipulation, however gentle, will give rise to pain, or by touching with the blunt end of a pin, which gives the sensation of a prick or "as if a bruise were being touched." The former method is the better to use on the front and sides of the chest, except in the case of women. Referred pain is frequently accompanied by headache, and areas of superficial tenderness on the trunk are often associated with similar areas on the scalp. The higher the area of tenderness on the trunk the further forwards will be the associated tenderness on the forehead; the nearer the tenderness on the abdomen approaches the tenth dorsal area, extending from the loin behind to the umbilical region in front, the nearer the tenderness of the scalp will approximate to the occipital region.

According to Dr. Head, referred pain is present when nodules of tuberculous infiltration are scattered among relatively healthy tissue. It may also be observed in cases of acute bronchitis or in cases of phthisis with recurrent attacks of bronchial catarrh. Every referred pain, when sufficiently intense, passes over and affects the same areas on the opposite side, but remains more marked on the side on which it began. On the whole the referred pain and superficial tenderness appear on the same side of the body and scalp as the lesion in the lung. It should be borne in mind that "the innervation of the lung is connected with the third and fourth cervical segmental areas, and with all the dorsal segmental areas from the third to the ninth. The lower lobe of the lung is particularly connected with the dorsal areas, especially from the fifth dorsal downwards to the ninth." The latter area, it may be remembered, stretches anteriorly from the nipple level to the umbilicus. The third and fourth cervical areas are intimately associated with apical affections.

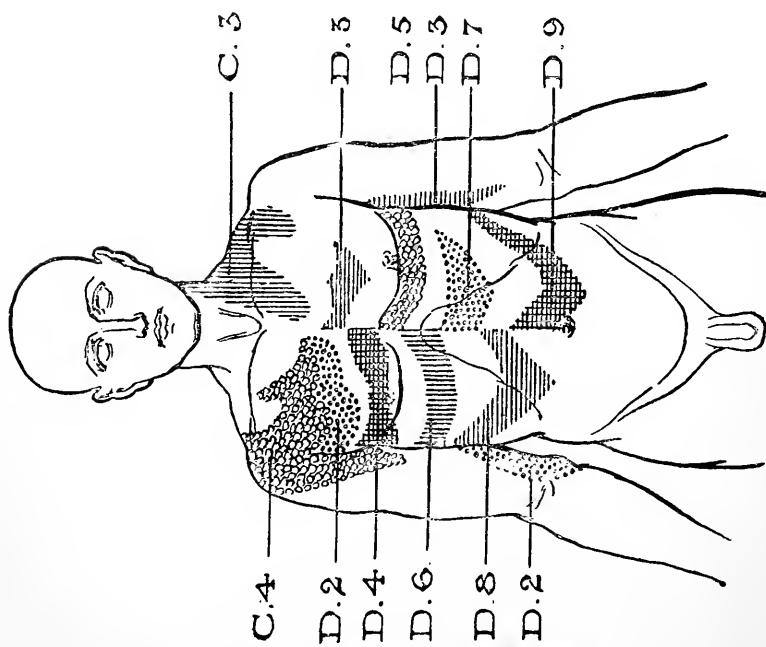


FIG. 12.

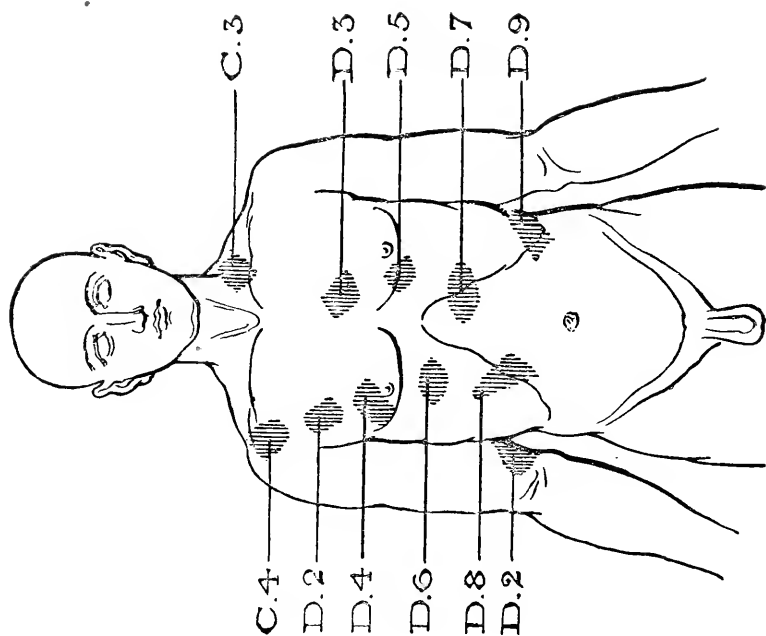


FIG. 13.

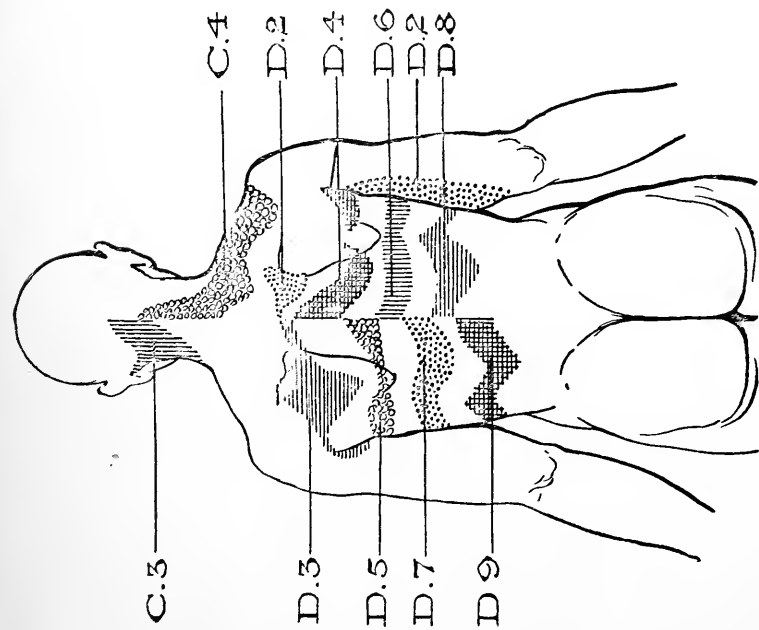


FIG. 14

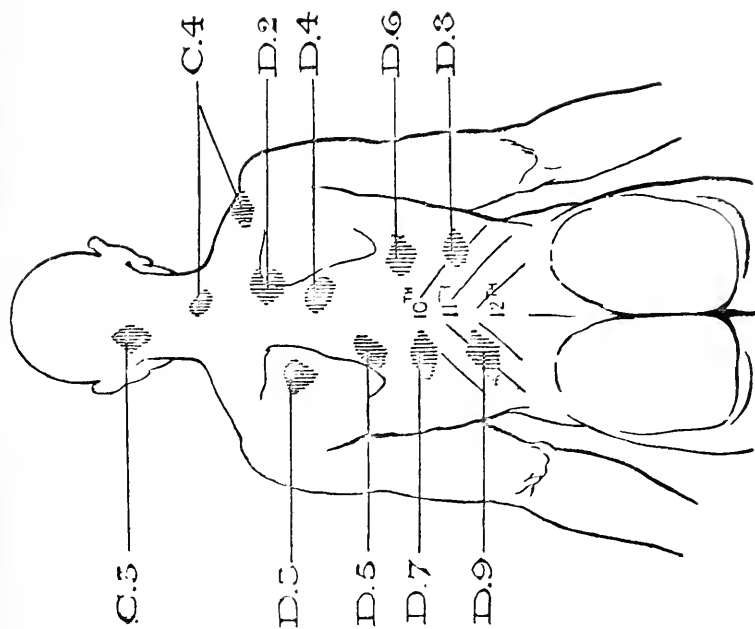


FIG. 15

The areas shown on Figs. 12 and 14 correspond to the distribution of certain afferent fibres that enter the third and fourth cervical and second to ninth dorsal posterior root ganglia. They constitute the regions to which pain is referred and within which tenderness makes its appearance in diseases of the heart and lungs. Tenderness may appear over any group of these areas in disease of the lungs, but the nearer the lesion is to the apex the more the cervical and upper areas of the thorax will be involved. In cardiac disease where the ventricle is principally affected, the tenderness will lie mainly over the upper areas of the chest (dorsal 2, 3, 4, 5); but where presumably the auricle is exposed to increased strain, tenderness makes its appearance within one or more of the areas from the fifth to the ninth dorsal (see pp. 122 and 342).

The tenderness that may appear as a direct consequence of pulmonary diseases lies over a territory comprising the following areas on Figs. 12 and 14: Cerv. 3 and 4, Dorsal 3 to Dorsal 9 inclusive. The more nearly the disease process involves the base of the lung the lower will be the superficial area of tenderness. The patient refers his pain to various points within this territory, and of these the most noteworthy are shown on Figs. 13 and 15. With these are associated areas of tenderness on the scalp. It must not be supposed that an isolated area in its complete limitation, whether in the trunk or scalp, can be marked out, excepting under circumstances of great rarity. For practical purposes it is only necessary to determine whether the tenderness that is present lies within one or more of the areas specified.

Dr. Head has recently drawn attention to "certain mental changes that accompany visceral disease" (Goulstonian Lectures, 1901), pointing out that these altered mental states are almost invariably found in association with referred pain. Local pain is not usually accompanied with these mental states, and the reflected pain must be of considerable intensity and some duration. The greater the area involved in pain and superficial tenderness the more certainly will mental changes make their appearance. The mental changes observed in connection with diseases of the respiratory organs consist of hallucinations of sight, hearing, and smell, and states of depression or sense of ill-being, exaltation or sense of well-being, and causeless suspicion.¹ Sometimes there is found to be also loss of memory and of power of attention, but these mental changes are not associated with reflected pain only. The "*spes phthisica*" or hopefulness so often observed in consumptive invalids is a mental condition of a more permanent character and of a different origin and nature from the mental changes here referred to. It arises from a failure to comprehend the gravity of the condition, the result of ignorance of the extent and progress of the disease.

¹ The Editor is much indebted to Dr. Head for the revision of this section, and also for Figures 12, 13, 14, and 15.

PHYSICAL EXAMINATION

This is of paramount importance in the diagnosis of diseases of the respiratory organs. The means employed in conducting the examination are inspection, mensuration, palpation, percussion, and auscultation. Each of these methods will be considered in detail.

INSPECTION

The method of inspection is that which should in all cases be adopted in the first instance. We note the general appearance, whether the patient looks well or ill, placid or anxious; the state of nutrition, whether wasted or well covered; the condition of the tongue, whether clean or coated; the attitude or decubitus, whether lying on the back or side, or sitting up in bed propped up by pillows; the presence or absence of anæmia, cyanosis, œdema, skin eruptions, clubbing of the fingers, venous engorgement or venous pulsation in the neck, dilatation of veins over the thorax, enlargement of glands, or scars or other abnormalities of the integuments. We observe particularly the character, rate, and rhythm of the respiration, whether it is quiet, easy, and regular, or hurried, laboured, and irregular. We note whether the alæ nasi are working, and whether the breathing is through the mouth or the nose.

SHAPE OF THE CHEST.—Although among normal healthy individuals the shape of the chest presents considerable variation, on the whole it conforms to one main type.

The ideal chest should be symmetrical on the two sides, well expanded, with its anterior wall slightly arched forward, and the sternum and vertebral column erect. The transverse section should be oval, with the wider diameter from side to side. The chest of the healthy male adult is somewhat of the shape of a truncated cone; the subcostal angle is about a right angle; and the widest diameter is at the level of the eighth rib, or between the seventh and eighth. In the female the bones are more delicate; the sternum is shorter and the subcostal angle is smaller, while the lower part is somewhat drawn in, so that the chest is more barrel-shaped than conical.

In the infant the lungs and the chest are relatively small; the thorax is short and conical; the spine is straight; the ribs are more nearly horizontal, and the cross-section is more circular than in later life. In the old, on the other hand, the ribs are more oblique, the curve of the spine is exaggerated, and the movements are more restricted.

The shape of the chest and its movements are affected by position.

In the recumbent position the dorsal curve of the spine is diminished, the upper ribs are elevated, and the abdominal viscera press backwards on the diaphragm. This position, therefore, diminishes the respiratory capacity, and is less advantageous for inspiratory movements. On this account, when dyspnœa is severe the patient can only breathe in a sitting position or propped up in bed with pillows, to which condition the term "orthopnœa" is applied.

Variations from the normal shape of the chest are numerous, but there are certain types which have been long recognised, and which merit special mention. What has been called the *phthinoid chest* is generally associated with small lungs and poor physique, and not infrequently with pulmonary tuberculosis. It is long and narrow; the ribs are more oblique than usual, and may reach or even overlap the iliac crests. The lower ribs are closer together and may actually be in contact, and the subcostal angle is very acute. The manubrium sterni is depressed and the angle of Louis (angulus Ludovici) is well marked. The shoulders droop, and the inferior angles of the scapulæ are tilted outwards, while the vertebral borders stand out like wings; hence the term *alar* is sometimes applied. A variety of the phthinoid chest is the *flat chest*, in which there is marked diminution of the antero-posterior diameter, and the costal cartilages instead of being convex forwards are flat or concave.

Another important and well-defined type is that known as *pigeon breast* (pectus carinatum). The sternum is unusually prominent, projecting like a keel; the lateral regions are flattened; and the cross-section of the chest is rather triangular than oval. This deformity points to the existence of some obstruction to free respiration during the early period of life when the ribs are soft. Another type is the *rickety chest*, in which there is a general drawing in of the sides of the chest and prominence of the anterior part, so that a furrow is formed on each side of the sternum, corresponding to the junctions of the ribs and costal cartilages. Still another type is that in which on each side there is a transverse furrow, called *Harrison's sulcus*, passing outwards and slightly downwards from the lower end of the sternum as far as the mid-axillary line. This furrow, which is not uncommonly present in a slight degree in normal cases, results from sinking in of the lower parts of the chest with inspiration, combined with the resistance offered by the abdominal viscera, and when well marked points to the existence of some chronic obstruction to the free entry of air into the lungs, as in cases of emphysema and chronic dyspnœa.

In what is known as the *cobbler's chest* there is a deep depression in the position of the lower part of the sternum. In shoemakers and some other artisans it results from continued pressure on the lower end of the sternum. But it may be found in an extreme degree, dating from early childhood, without the operation of any such cause.

In women the shape of the chest may be much distorted by *tight lacing*. In a typical case of this deformity the diameter is greatest at the level of the fourth rib, below which it diminishes, at first gradually, then after the sixth rib more markedly. The ribs below the fourth are more oblique and the costal angle is acute, 40° in extreme cases. The greatest constriction of the stays is over the level of the distal end of the ninth rib a little above the umbilicus. The result of the pressure is to raise the arch of the diaphragm and so diminish the capacity of the chest, especially of the lower part, and also to impede the movement of the lower ribs.

The chest is sometimes extremely mis-shapen as the result of *angular curvature*. *Kyphosis*, which is an exaggeration of the normal thoracic curve, produces a type of chest closely resembling that of emphysema. *Lateral curvature*, or *scoliosis*, also produces deformities of the chest, which, however, are usually asymmetrical. It must be borne in mind that sometimes the scoliosis is itself the result of unilateral contraction of the chest, due to disease of the lung or pleura.

The alterations of shape so far considered more often point to the existence of past than indicate present disease. The changes of shape which commonly point to present disease are local, unilateral, or bilateral enlargement, or general or local contraction. Unilateral enlargement may be due to the presence of air or fluid in the pleura, or to new growth or hydatid in the lung; but the effusion must be great or the tumour large to bring this about, as the retraction of lung and displacement of the other organs will make room for a considerable addition to the contents of the thorax without necessitating any bulging of the chest-wall. Bilateral enlargement results from the hypertrophous form of emphysema, and a description of the *barrel-shaped chest* characteristic of this disease is given on p. 184. Local contraction is much more common than local enlargement, and usually is caused by pulmonary collapse, or excavation, or fibrosis of the lung. The contraction is sometimes very marked when the lung does not expand after the absorption of a pleural effusion. There is then drooping of the shoulder on the affected side, diminished circumference and narrowing of the intercostal spaces, and alteration in the relative position of the soft parts such as the nipple. Deformities of all kinds are more likely to occur in the case of children, in whom the ribs are much more yielding than in adults.

The position of the heart should be carefully noted as far as it can be made out by inspection, and later by palpation. The heart is displaced towards the sound side by liquid or air in the pleura, downwards by emphysematous lung, and towards the affected side in cases of local contraction such as excavation and fibrosis. Pulsation in such cases will be perceptible in abnormal positions.

Inspection of the shape of the chest should be followed up by

MENSURATION. The circumference of the chest is an index of the thoracic development, and in full inspiration its mean measurement is 35 inches, rather more than half the height. The two sides of the chest may be compared by means of the measuring-tape. The diameters may be taken by means of calipers. The diameter from side to side averages in men from 10 to $10\frac{1}{2}$ inches, and in women from $9\frac{1}{2}$ to $9\frac{3}{4}$ inches at the nipple level, while the antero-posterior diameter averages $7\frac{1}{2}$ inches.

In cases where there is deformity it is, however, more useful to employ the cyrtometer, an instrument consisting of two pieces of flexible lead piping, hinged together by a piece of rubber tubing. The lead piping can be moulded to the form of the chest, then removed without disturbing its shape, and an exact tracing can be taken on a sheet of paper, and a permanent record thus obtained.

In addition to observing the shape of the chest, we also study by inspection and measurement **THE MOVEMENTS OF RESPIRATION.** In men, the diaphragm being the principal agent of respiration, the abdominal movements are the more marked; while in women and children, in whom the intercostals play a more prominent part, the movements of the chest, especially in its upper part, are the more conspicuous. The extent of movement of the chest-wall may be estimated by measuring with tape the circumference at the nipple level at the end of expiration and at the end of deep inspiration respectively. The difference in the circumference will be found to amount to $2\frac{1}{2}$ or $3\frac{1}{2}$ inches in the normal adult. In emphysema both inspection and measurement show diminished movement on both sides. It should be observed whether the movements on the two sides are equal, for inequality of movement usually points to the existence of some physical defect on the side where the movement is the less. An inequality which may be little noticeable when the breathing is quiet may become conspicuous when a full inspiration is taken. The movements should be inspected not only from the front, but also from above, which can be conveniently managed if the patient is seated and the examiner stands behind.

In large pleural effusions and pneumothorax there is little or no movement of the whole of the affected side. When there is collapse, or pulmonary consolidation, or excavation, there is diminished movement of the overlying chest-wall. Sometimes over a cavity the chest-wall sinks in during inspiration and bulges outwards in expiration. Movements are generally diminished when they are productive of pain, as in pleurisy. When there is obstruction of the larynx or trachea, so that air cannot freely enter the lungs, the lower ribs are drawn inwards during inspiration, and this may be extremely marked in the case of children whose ribs are yielding. When air does not enter freely into the upper lobes, the movements of the lower part of the chest are exaggerated, and *vice versa*.

PALPATION

By means of palpation we can recognise the presence of local tenderness, judge of the extent and rate of the movements, estimate the amount of resistance, appreciate the vibration of the chest-wall or vocal fremitus produced by the spoken voice, and detect the existence of pulsation or fluctuation. The results of inspection of the movements can thus be confirmed by placing the hands on the chest-wall below the clavicles, and the movements of the two sides may sometimes be better compared in this way than by inspection. The resistance may be estimated and the presence of tenderness may be revealed by pressure with the fingers.

The *vocal fremitus* is felt by placing the hand flat on the chest and getting the patient to repeat, in a loud voice, "Nine hundred and ninety-nine," or some such phrase. Corresponding parts of the two sides should be carefully compared. Vocal fremitus depends on the fact that the voice sounds are capable of throwing into vibration the tissues which conduct them, which vibration is communicated from the chest-wall to the hand.

Deep, sonorous tones set up more vibration than those of higher pitch and less volume. Hence vocal fremitus is always more marked in the case of a man than in a woman or a child. For this reason the patient should be asked to speak in the lower tones of his register. When the lungs are healthy the fremitus may be felt wherever they are in contact with the chest-wall, and is almost equal on the two sides, but it is more marked nearer to the larynx, as over the front of the chest, than farther away, as at the base. The fremitus is increased when the conducting power of the lung is greater, as it is in consolidation and over cavities. Vibration travels badly from one medium to another, as from the lung to liquid or air; and accordingly the fremitus is diminished when there is serum, pus, or air in the pleural cavity, thickening of the pleura, or a considerable layer of fat or oedematous tissue in the thoracic wall. Fremitus may be felt when the patient coughs (*tussive fremitus*), a fact which it is useful to remember in the examination of children.

The vibrations which give rise to audible rhonchi (*bronchial fremitus*) may be felt in the same way as those of the voice, and the grating or scraping of one layer of pleura on the other (*pleural fremitus*) may also be felt as well as heard.

The existence of pulsation in abnormal situations has been referred to in connection with inspection. It can, however, be more readily and with greater certainty detected by means of palpation. It has been explained that such pulsation is mostly due to displacement or uncovering of the heart. Pulsation may very exceptionally be communicated to fluid in the pleural sac. Pulsation has generally, but not

exclusively, been observed in the case of purulent effusions on the left side (*pulsating emphyema*), and usually in the intercostal spaces near the sternum or to the left of the apex of the heart. In cases of large pleural effusions, especially in children, a fluid thrill or sense of fluctuation may occasionally be felt by placing one hand in front of the thorax and tapping with the other behind, or *vice versâ*.

PERCUSSION

Examination by means of percussion consists in listening to the sound elicited by striking, *secundum artem*, the surface of the chest so as to obtain information as to the conditions within. Percussion may be performed either by striking with the tips of one or more fingers or with a small hammer (called a "plessor"). It may be applied either directly to the surface of the chest or to one of the fingers of the other hand, or, when a plessor is used, to a small flat plate of bone or other material (called a "pleximeter") applied closely to the surface. When percussion is done without any intermediate substance, it is called "immediate"; when otherwise, it is called "mediate." The immediate method is seldom employed, as the mediate method gives better results and is less unpleasant to the patient. There is a distinct advantage in using the fingers rather than a plessor and pleximeter, inasmuch as thereby one is able to appreciate by the sense of touch the degree of resistance, as well as the vibrations produced by percussion.

The sound produced by percussion takes its character principally from the physical condition of the lung or other substance which lies beneath the chest-wall, but it is modified by the degree of thickness, elasticity, etc., of the parietes. The character of the sound depends on whether free vibrations can be set up or not, in the part percussed. The term "resonance" is applied to sounds produced by percussion of parts which vibrate freely, such as the chest-wall over normal lung or over a pneumothorax or large cavity; while the term "dulness" is applied when the vibrations are damped, as when there is a pleural effusion, or solid lung, or a tumour beneath the part percussed. The difference between resonance and dulness may be readily learnt by percussing over the normal lung and over the fleshy part of the thigh respectively. It is necessary to be acquainted with the limits of normal pulmonary resonance. In front, on the right side there is resonance from the apex to the sixth rib, where the liver dulness commences. On the left side the pulmonary resonance reaches as low as the fourth costal cartilage, where the cardiac dulness begins. The limits of cardiac dulness are the mid sternal line on the right, and on the left a line slightly convex outwards, passing from the sternal end of the fourth costal cartilage to the apex of the heart. Below, the pulmonary resonance is continuous with the resonance due to the stomach, which may be distinguished by

its different character. The thoracic portion of the stomach resonance occupies a semilunar area, called Traube's space, about $3\frac{1}{4}$ inches wide where it is broadest, lying along the anterior border of the ribs, and reaching on the left to the eighth or ninth rib. Where the lungs are enlarged, as in hypertrophous emphysema, the areas of cardiac and hepatic dullness and of Traube's space are diminished or altogether obliterated. Traube's space is also diminished in left pleural effusion, while it is enlarged when the lung is atrophied, as in the senile form of emphysema. The pulmonary resonance extends posteriorly down to the tenth or eleventh rib on both sides; in the right axillary region to the eighth rib, and in the left to the ninth rib. The note posteriorly, especially in the supra and infra-spinous regions, is less resonant than that in front. A careful comparison of the two sides is necessary to recognise the slighter degrees of dullness.

The percussion note is more resonant when the parietes are thin, there being little subcutaneous fat and poor muscular development. Resonance is increased when the lungs are over-expanded, as in emphysema. If on percussion of the chest we get a dull sound in the region of normal pulmonary resonance, we are justified in drawing the conclusion that there is a diminution of the normal air-containing tissue, either fluid, thickening of the pleura, consolidated lung, or solid tissue of some other kind underlying the part percussed. Between dullness and good resonance there are various intermediate degrees, and it is highly important to compare one side with the other, for the recognition of slight degrees of dullness may be of great importance in diagnosis. Besides noting whether the percussion sound is dull or resonant, we observe its intensity, pitch, quality, and duration. In the case of a superficial cavity in the lung of a certain size the percussion sound acquires definite pitch and tone, and this is due to the fact that the air contained in a cavity is thrown into vibration in a definite manner depending on the form and size of the cavity. The pitch is raised when the mouth is held open during percussion, lowered when the mouth and nostrils are closed. The note obtained by percussion over a cavity has been called *cavernous*, or when the cavity is large *amphoric*. The term *tympanitic* has also been applied in this sense, but its use has led to some confusion, as it should properly denote the low-pitched full sound produced by percussion of the abdomen when the intestines are distended. The note over a cavity is similar to that obtained over an undistended knuckle of intestine or stomach, and does not at all resemble the drum-like sound obtained over a tympanitic or distended abdomen. A somewhat similar note is sometimes observed over the upper part of the lung when the lower part is compressed by fluid (so-called *Skodaic resonance*).

In the case of a cavity the sound also sometimes acquires a peculiar quality called *bruit de pot fêlé*, or "cracked-pot sound," which is a

chinking or hissing noise like that obtained by striking the knee with the clasped hands. For the production of this sound the cavity should be of fair size, superficially situated, and communicating with a bronchus; the patient's mouth should be open, and the percussion should be sudden and forcible. The cracked-pot sound is due to the sudden expulsion of some of the air from the cavity. It is not an absolute sign of a cavity, for it may be obtained over the upper part of the healthy chest when the walls are yielding, as in the case of young children.

The following are some of the different kinds of percussion sound which have been recognised:—Increased resonance, such as the sound audible over an emphysematous lung; normal resonance, the sound audible over normal lung; cavernous or amphoric resonance, a sound possessed of a definite pitch, depending on the size of the cavity and sometimes altered by opening or closing the mouth, and a hollow quality; tympanitic resonance, a term sometimes applied to cavernous resonance, and sometimes to a sound similar to that obtained by percussing a tympanitic abdomen; Skodaic resonance, the high-pitched sound obtained by percussing the upper part of the lung when the lower part is compressed by pleural effusion; diminished resonance in various degrees up to absolute dulness; cracked-pot sound or *bruit de pot fêlé*.

Percussion resistance.—One of the chief advantages of percussing with the fingers alone is that it enables us to appreciate the degree of resistance offered by the chest-wall to percussion. This resistance may be altered by changes either in the chest-wall itself, or in the contents of the thoracic cavity. Unusual thickness of the bony framework, close proximity of the ribs, brawny muscles, or a deep layer of subcutaneous fat will produce a sense of greater resistance, and such causes will be readily recognised. The resistance will be increased when there is consolidation of lung in proportion to its completeness, still more in pleural effusions and in cases of thickened pleura, but most of all in solid growths of the lung or pleura. Diminution of resistance is both less common and less important, and may occur when the bones are thin, the intercostal spaces wide, or the chest poorly covered, and in cases of emphysema or pneumothorax.

AUSCULTATION

This consists in listening to the sounds produced in respiration, speaking, whispering, coughing, etc. The more important of these sounds are only audible when the ear is directly applied to the bare or covered chest, or when a special instrument called a stethoscope is used to conduct the sound from the uncovered chest to the ear. It is unnecessary to describe the different forms of stethoscope. The two chief forms are the wooden single stethoscope and the binaural stethoscope, with the use of both of which it is well to be familiar. Some sounds

are better conducted by the wooden stethoscope, but for general purposes the binaural is the more convenient.

The sounds produced in respiration which are audible on listening over the lungs are due to the movements of air through the air-passages, or to the rubbing of the two layers of pleura on one another when roughened by inflammatory exudation. The character and origin of the normal breath sounds have been discussed at p. 12. From a variety of causes these sounds undergo modifications in disease, and with the nature and meaning of these modifications it is of the highest importance to be acquainted. From the character of the breath sounds we learn much as to the condition of the lung itself.

CHANGES IN THE NORMAL PULMONARY BREATH SOUNDS.—Vesicular breath sounds are generally audible over the whole thorax, but are most distinct where the chest is most thinly covered. They are weak where there is a thin layer of lung, as at the apex and along the anterior borders. Wherever normal vesicular breath sounds are audible the inference is justified that the lung tissue beneath is permeable to air. In *exaggerated breathing* the inspiratory sound is louder and harsher than normal. This is sometimes met with over one lung when air does not enter the other freely. In children the breath sounds are normally exaggerated, and are spoken of as *puerile*, and the probable causes are the thinness of the chest-walls and the greater elasticity of the lungs. The breath sounds may be *diminished*, as in the case of emphysema, where respiration is shallower than normal; or they may be *suppressed*, as when the lung is collapsed from the pressure of air or fluid in the pleura, or compressed by tumours, or by an enlarged heart or pericardial effusion, or when a bronchus is blocked by secretion, growth, or a foreign body. The sounds of inspiration and expiration normally succeed each other so closely as to be almost continuous. Under certain conditions, such as advanced emphysema, however, they are divided by a distinct pause (*divided respiration*). Divided respiration is a feature of "bronchial breathing." Sometimes the breath sounds have a *wavy, jerky, or interrupted* character, to which, when well pronounced, the term *cog-wheel breathing* has been applied. The inspiration is not continuous, but is broken up into a number of short waves. Sometimes this is a nervous phenomenon, the patient inspiring slowly and shallowly, and air entering successively into different parts and not all at once, in which case a quick, deep inspiration will cause it to disappear. Sometimes, however, it depends on tuberculous infiltration, which interferes with the regular and even entrance of air into the affected part; and in such cases, if removed by deep inspiration or coughing, it tends to return.

Prolonged or harsh expiration is generally the result of some obstacle to the free escape of inspired air, and is met with in cases of bronchial catarrh and emphysema.

Bronchial or tubular breathing may be defined as breath sounds of

similar character to those audible over the trachea, in which the inspiration and expiration are of equal length and intensity, are of a distinctly blowing character, and are separated by a short interval, but differing inasmuch as the expiratory sound is usually the louder and harsher. Some make a distinction between bronchial breathing and tubular breathing, defining the latter as being more whiffing and of higher pitch than the former, but for all practical purposes the two are identical. In different cases the pitch and intensity vary greatly. Bronchial or tubular breathing is audible on listening over the chest only in certain pathological conditions, such as consolidated lung, cavities, and collapsed lung when it is in contact with the chest-wall.

Consolidation of the lung acts in two ways in modifying the breath sounds: as no air enters the affected part, the alveolar portion of the inspiratory murmur is suppressed, while, the alveoli being filled with exudation, the dissipation of sound, which resulted from the free communication of the tubes with the alveoli, is prevented, conduction is much more perfect, and the glottic sounds are almost as well conducted to the ear as they are by the trachea. It accordingly comes about that "bronchial" or "tubular" breathing is usually audible over consolidated lung. If the tubes as well as the alveoli are blocked, then if tubular breathing be audible at all it is very faint. When the lung is collapsed no air enters it, but the larger bronchi are brought nearer to the surface; tubular breathing of greater or less intensity may be audible, or the breath sounds may be entirely suppressed.

Cavernous and amphoric breathing.—When a cavity communicating freely with a bronchus is present, the cavity walls prevent the diffusion of sounds conducted from the bronchus, which are conducted with increased intensity and modified by resonance in the cavity, which imparts to them a distinctive character like that resulting from blowing over a bottle or jar. According to the degree in which they possess this quality they are called *cavernous* or *amphoric breath sounds*. The quality is sometimes metallic, but more often is like that produced by blowing over the mouth of a jar. It is only when the cavity is of a considerable size and superficial, and when it freely communicates with a bronchus, that the amphoric quality is well marked. Amphoric breathing is sometimes audible over a pneumothorax, but is not so common as suppression of the breath sounds.

Broncho-vesicular or indeterminate is a term which has been applied to breath sounds which are neither bronchial nor vesicular but a kind of combination of the two. This type of sound may normally be heard over the manubrium sterni and posteriorly between the spines of the scapulæ, and there arises from the mingling of the conducted tracheal and bronchial breath sounds with the pulmonary. When audible over abnormal areas it indicates diminished entry of air into the vesicles.

Post-tussive suction sound.—Sometimes, after the patient coughs

and then takes a deep breath, air may be drawn into a cavity with sufficient force to give rise to a kind of hissing or suction sound. This post-tussive suction sound is not often heard, but when present it is a very sure sign of a cavity.

Stridor is a peculiar, harsh, vibrating, noisy, but somewhat musical sound accompanying inspiration and generally audible at some distance from the patient. It is due to laryngeal or tracheal stenosis.

THE AUSCULTATION OF THE VOICE SOUNDS.—The conduction of the spoken and whispered voice sounds follows the same laws as that of the glottic breath sounds. The spoken voice sounds are produced at the larynx, and are modified by resonance in the buccal, nasal, and pharyngeal cavities. The whispered sounds which originate in the lips and tongue have been found experimentally to be exceedingly well conducted by means of tubes, and they are well conducted to the chest-wall in all conditions which promote the conduction of the glottic breath sounds. When the whispered voice is distinctly audible on auscultation we call it *pectoriloquy*. This occurs over consolidated lung, and sometimes over compressed lung, while over a cavity or pneumothorax the whispered voice may possess a superadded cavernous quality.

The conduction of the spoken voice is a little different. We have seen when discussing palpation how the vibrations of the voice are in normal conditions transmitted to the surface of the chest. They are of much greater intensity than the breath sounds, and thus are perceptible at a greater distance. Ordinarily we simply hear the noise of the voice, not the articulate utterance. The same conditions which diminish or increase the vocal fremitus will diminish or increase the vocal resonance. *Increased vocal resonance* we call *bronchophony*, and it generally goes along with pectoriloquy and bronchial breathing. Over a large cavity the spoken voice usually has a cavernous or amphoric quality from resonance in the cavity.

Ægophony.—In the case of a pleural effusion of moderate amount it will be observed that at the upper level of the fluid posteriorly the voice sound has a peculiar bleating quality. The term *ægophony* has been applied to the sound from its fancied resemblance to the cry of a goat. It is like the voice as heard through a telephone, or as uttered through a comb with a piece of paper in front of it. The explanation of *ægophony* which is generally accepted is that the lower tones of the voice are not well transmitted by the fluid. The ordinary spoken voice is made up of fundamental tones and their harmonics. Low tones travel from air to liquid less well than higher tones, and accordingly a sound composed of a fundamental tone and its harmonics is altered on passing through a layer of liquid by the deadening of the fundamental, while the harmonics become relatively louder. Sometimes, however, *ægophony* is observed over consolidated lung without the presence of fluid, on which, therefore, the sound cannot be altogether dependent for its production.

ADVENTITIOUS SOUNDS may accompany the respiratory sounds under a variety of different conditions, and may be produced in various ways. The rubbing of the layers of pleura when roughened by exudation will produce a friction sound. When secretions are present in the trachea, bronchi, or alveoli, or in pulmonary cavities, sounds may be produced by air bubbling through them, or when they are thick and viscous by passing through the narrowings in the channels they cause. The sticking together of the mucous membranes lining the bronchiole walls during expiration, and their separation in inspiration, is also capable of giving rise to certain sounds. The agitation of fluid in air-containing cavities will likewise produce characteristic noises.

Before discussing the different kinds of adventitious sounds, it must be pointed out that there is considerable want of uniformity among physicians as to the names given to the various sounds. The terms here employed are those in most general use at the present time.

Pleuritic friction sound.—The mode of origin of this sound admits of no dispute, being clearly the rubbing of the roughened surfaces of the visceral and parietal layers of the pleura. It is of varying intensity, and in different cases is described as rubbing, grating, or creaking. It may be heard throughout both inspiration and expiration, or only at the height of inspiration and the beginning of expiration. As a rule it is not conducted to any great distance from its place of origin. It is sometimes intensified by pressure with the stethoscope, which possibly acts by bringing the roughened surfaces into closer contact. We have already pointed out that it is sometimes possible to feel the friction as well as to hear it. It is sometimes difficult to distinguish certain sounds produced within the lungs from friction sounds. Sounds of pulmonary origin are, however, usually altered by coughing, and are not affected by pressure with the stethoscope. Friction in the pleura overlying the heart (pleuro-pericardial friction) may closely simulate true pericardial friction, but it may be distinguished in some cases by the fact that it is suspended by temporarily holding the breath after taking a deep inspiration, or at the end of expiration. Creaking sounds of pleural origin but closely resembling râles are sometimes due to the presence of partial adhesions or local irregularity or thickening of the pleural surfaces.

Rhonchi.—When the lumen of one of the larger tubes is narrowed by the presence in it of a mass of mucus or thick secretion or in other ways, a sound is likely to be produced by the passage of air backwards and forwards in inspiration and expiration. A snoring or whistling essentially somewhat musical sound may arise in this way, to which the name of *rhonchus* has been given. The significance of rhonchus is the presence of secretion in the larger and medium-sized tubes, or swelling of the mucous membrane, and it is met with principally in cases of bronchitis. As rhonchi are produced in the trachea and bronchi, they

are generally audible over a wide area of the chest-wall. The low-pitched and snoring variety which is called *sonorous rhonchus* has its origin in the larger tubes, and can often be removed by dislodging the secretion. The whistling, high-pitched variety which is called the *sibilant rhonchus*, or simply *sibilus*, is due to the presence of secretion in, or swelling of, the mucous membrane of the medium-sized tubes, and generally is unaltered on coughing.

Râles.—The term *râle*, which literally means rattle, is applied to the sound produced in the smaller tubes by air passing through secretion, or by the separation of adhering surfaces by drawn-in air.

The classification of *râles* is a matter of some difficulty, and a very large number of different kinds have been named and described. For practical purposes we may divide them into the *moist or bubbling* and the *dry or crackling*. These again may be divided into fine, medium, or coarse, according to their quality or loudness. Bubbling *râles* arise from the passage of bubbles of air through fluid; the crackling *râles* suggest an origin in the separation of surfaces rendered sticky by thick and tenacious secretion. *Râles* may be audible in inspiration alone, or in expiration alone, or in both. In cases where they are due to secretion in the finer bronchi, they are most commonly heard at the height of inspiration, and just at the beginning of expiration. When due to secretion in the larger tubes, they are audible both in inspiration and expiration. *Râles* may be few or many, giving one the impression of only a few bubbles or the combination of a large number. They are abundant when there is much secretion and free entry of air to the affected part. As regards loudness of the *râles*, it is greater when there is much secretion, the breathing deep, the bronchi of wide lumen, and the part affected near the surface. *Râles* follow the same laws, as regards conduction, as other sounds, and are badly conducted by normal lung from their place of origin. If *râles* of nearly equal intensity be audible over a wide area, and the lung be not consolidated, the cause of their production must also be in operation over a wide area. *Râles* are badly transmitted from one side to the other, and when *râles* are heard on both sides it usually points to the presence of disease on both sides.

It is a very important matter for the student to appreciate the differences in the quality of *râles* when heard over consolidated lung, over cavities, and over normal air-containing lung tissue. Over consolidated lung, *râles* have a bright or clear character, which was believed by Skoda to be imparted to them by resonance or, as he called it, consonance in the bronchial tubes. On this account the term *consonating râle* was applied. It is really the mingling of the *râle* with the conducted tracheal breath sounds which gives it its peculiar character. What is called the *crepitant râle* or *crepitation* is a sound which has been aptly compared to that produced by rubbing the hair

between the fingers close to the ear. It is due to the entrance of air into alveoli which are partly filled with secretion, and occurs mainly at the end of inspiration.

Metallic tinkling.—Râles audible over a cavity by resonance in the cavity may acquire a cavernous or even metallic or tinkling quality. The typical "metallic tinkling" sound is only heard over large and superficial cavities or over a pneumothorax. It resembles the sound produced by letting a drop of water fall into a hollow metal jar. It may be due in some cases to a drop falling from the upper to the lower surface of a cavity, but more commonly it results from the bursting of a bubble in its interior.

AUSCULTATION OF COUGH.—In conducting a systematic examination of the respiratory system it is important to auscultate during as well as after coughing. The cough itself, like the voice sounds, is better conducted through consolidated tissue, while over cavities it has a hollow or metallic quality. More important, however, is the fact that coughing may entirely alter the character of the breath sounds, or may bring out râles till then unheard. Coughing may dislodge plugs of mucus, and thus tubular breathing or normal breathing may be heard where previously the breath sounds were weak. Coughing shakes up the secretions in cavities and bronchi, and thus produces characteristic sounds, gurgling, bubbling, or crackling. The deep inspiration which follows a cough is sometimes accompanied with râles, when none have been heard before. After coughing, the situation of râles may be altered, due to the displacement of the secretion.

AUSCULTATORY PERCUSSION.—Combined auscultation and percussion are sometimes useful in determining the limits of a pulmonary cavity or a pneumothorax. The principle of the method consists in recognising the change in the percussion sound on passing beyond the boundary of a cavity or organ over which we are listening. In the case of a cavity the stethoscope is placed over its centre and light percussion is made in the neighbourhood, and the character of the sound noted. We then percuss in the same way at a distance, and gradually approach until we hear a sound of the same character as at first. This shows us that we have reached the boundary of the cavity, which may be mapped out by this means. Some believe that they are able to map out the lobes of the lungs in this way. Another method of employing auscultatory percussion is to percuss the chest, and at the same time listen over the patient's open mouth. This method sometimes enables us more clearly to recognise the cracked-pot sound or cavernous percussion sound. The sound is conducted from the cavity along the bronchi and trachea to the mouth.

Bruit d'airain.—A well-known sign obtained by auscultatory percussion is what is known as the *bruit d'airain*, bell sound or coin sound. It is a clear metallic sound audible under certain circumstances

when auscultation is combined with percussion by means of two coins, one being employed as plessor, the other as pleximeter. It is only produced in cases of pneumothorax and large superficial cavities. It is due to metallic resonance in the cavity, and both auscultation and percussion must be performed over the cavity.

Succussion sound.—When air and fluid are present in the pleural cavity, as in the case of hydro-pneumothorax or pyo-pneumothorax, a splashing sound may be heard on listening to the chest when the patient is shaken or moves quickly from one position to another. This sound was known to Hippocrates, and is sometimes called the Hippocratic succussion sound.

Cardio-pulmonary murmurs.—A brief reference may be made to certain cardiac murmurs, which are not uncommonly met with in cases of pulmonary disease, and are independent of cardiac lesions. One of the most frequent is a blowing systolic murmur, audible over or in the neighbourhood of a cavity. It is due to the sudden driving out of air from the cavity by the cardiac impulse, and accordingly is similar in origin to the *bruit de pot fêlé*. The air returns during diastole, but without sufficient force to produce a murmur. Another type of murmur is that where there is pressure on the pulmonary artery by the contraction of adhesions or fibrous bands. This gives rise to a systolic murmur loudest in the second left interspace. Similarly in cases of chronic apical induration there may be pressure on the subclavian artery producing a murmur in the subclavian region. The displacement of the heart following the absorption of a left pleural effusion results sometimes in a loud systolic murmur, which has been attributed to the expulsion of the air from the larger bronchi with each impact of the heart. A murmur of similar origin may be observed when the lung tissue adjacent to the heart is consolidated. Deformities of the chest and displacements of the heart from any cause may lead to the production of systolic murmurs.

The conduction of the heart sounds.—Sounds do not as a rule travel well from one medium to another, and thus the heart sounds are badly conducted by the air-containing normal lung from the solid walls of the heart. When, however, the lung is consolidated, its power of conducting the heart sounds is increased. We therefore find that the heart sounds are audible with increased intensity over a portion of consolidated lung which is in close relation with the heart. The heart sounds are also heard more loudly over a pulmonary cavity lying near the heart, and sometimes by resonance in the cavity acquire a cavernous or metallic quality. In the case of pneumothorax this phenomenon is not so common, probably because the collapsed lung separates the heart from direct contact with the pleural cavity. Over emphysematous lung or a pleural effusion the heart sounds are conducted with diminished intensity.

RADIOSCOPY

The method of examination by means of the Röntgen rays promises to be a very useful aid to investigation by other methods. It requires, however, great experience in the study of both normal and abnormal cases to properly interpret the appearances. In health the pulmonary image should be quite clear, from apex to base, with the exception of a few ill-defined, shadowy, nearly vertical lines to the right of the heart shadow, which appear to be caused by the opacity of the junction of the pericardium with the pleuræ. The movements of the diaphragm are well seen, and should be equal on the two sides, any difference of movement being generally significant of disease. In cases of tubercle of lung the movement of the diaphragm on the affected side is always much less than on the non-affected or less affected side. The depression of the diaphragm caused by pneumothorax is clearly revealed. Consolidation, tubercle, tumour, effusion, all cast a corresponding shadow. Pneumonic consolidation casts a dense homogeneous shadow, which does not, however, completely obliterate the shadows of the ribs. Caseous tubercle gives a definite, dense, flocculent shadow; gray and yellow tubercles cast less dense but still definite shadows. The shadow of a growth cannot, however, be distinguished from that of tuberculous or other consolidations, but the distribution of the shadow may help in the diagnosis. Thickened pleura or a thick layer of fibrinous exudation casts a decided shadow. A serous effusion throws a less dense shadow than one which is purulent. With serum the outline of the ribs is not obliterated, and no shadow at all may be visible in the case of a moderate effusion, while in the case of a well-marked empyema the shadow may be so dense that the outline of the ribs is quite lost. When there is effusion the upper margin of the shadow should be clearly defined. Radioscopy is very successful in revealing the presence of cavities in the midst of solid tissue, and their size can be determined much more exactly than by means of auscultation. In emphysema the lung appears more translucent than normal. The position of the heart as indicated by its shadow is sometimes a valuable aid to diagnosis. In doubtful cases a skiagram should be taken, as shadows may appear on it which may be missed by screen examination.

DISEASES OF THE LARYNX

ACUTE CATARRHAL LARYNGITIS

Symptoms.—In slight cases there is a feeling of dryness, heat, pricking, tickling, roughness, or actual soreness referred to the larynx, combined with huskiness, hoarseness, or aphonia. There is often an irritable cough with at first little or no expectoration, although later a little thick, viscid mucus, sometimes slightly streaked with blood, may be spat up. Secretion is never abundant unless there be associated tracheitis or bronchitis. In cases of a more severe type, in addition to there being alteration or loss of voice and cough, pain accompanied by local tenderness is felt over the larynx, there is some discomfort in swallowing, and constitutional disturbance shown by feverishness and malaise, absent in the slighter cases, may be present. There is seldom dyspnoea in the case of adults unless there be œdema of the larynx.

In children more severe, sometimes alarming, symptoms may arise. During the day, only the presence of coryza, slight cough, and hoarseness may be noticed, but at night the child wakes up with severe and urgent dyspnoea. There is then incessant cough, which, as well as the voice, is husky and harsh. The face, flushed at first, becomes pale and perhaps cyanotic. After a time the breathing becomes easier and the child falls asleep again, but the paroxysm is liable to return either on the same night or on several subsequent nights in succession. This condition has been called *laryngitis stridulosa* or *spurious croup*. The paroxysm probably depends on thick, viscid mucus collecting about the glottis, and at last interfering greatly with the respiration until finally it is dislodged.

Cases to which the term *laryngitis hæmorrhagica* has been applied are occasionally, but very rarely, met with. In these, somewhat profuse bleeding occurs from the larynx, generally after strain of the voice, violent cough, or retching.

Laryngoscopic appearances.—On laryngoscopic examinations, one or both vocal cords are seen to be reddened, either in part or as a whole. The colour may vary from a pale pink to a redness as deep as that of the rest of the larynx. Some viscid secretion will probably be seen clinging to the cords, which may

show imperfect tension on phonation. In severe forms, swelling as well as hyperæmia, both of the ventricular bands and of the cords, may be seen; and in exceptional cases small superficial symmetrically situated ulcers or erosions appear on the free margins of the cords. In the rare hæmorrhagic cases, the presence of streaks of blood or varicose veins may be recognised.

Treatment.—In the milder cases, complete rest to the voice, confinement to bed for a day or two in a well-ventilated room maintained at a comfortable temperature, and the use of soothing steam inhalations will soon effect a cure. A good form of soothing inhalation is a teaspoonful of compound tincture of benzoin added to a pint of water at 140° F. The inhalation of nascent chloride of ammonium is also useful. Soden mineral, or chloride of ammonium lozenges relieve the soreness of the throat. In more severe cases, the patient may be given ice to suck and cold compresses or ice poultices may be applied externally, but sometimes warm applications are more comforting. The benzoin inhalation may be frequently used, or the throat may be sprayed with a 5 per cent solution of menthol in liquid paraffin. Troublesome cough may be relieved by sipping warm milk, by sucking codeine or cocaine pastilles or cocaine and rhatany lozenges (B.P.), by using a menthol inhalation (20 drops of alcoholic solution (20 per cent) on an oronasal inhaler), or a linctus, or by small doses (gr. $\frac{1}{16}$ to $\frac{1}{12}$) of heroin hydrochloride. In cases where there is fever a saline aperient should be administered at the beginning of the illness and followed by a mixture containing two to three drachms of the solution of the acetate or citrate of ammonia to be taken three times a day. In the severe nocturnal attacks of dyspnœa occurring in children, a prompt emetic such as a teaspoonful of ipecacuanha wine repeated every ten minutes until vomiting is produced, and the local application over the larynx of hot compresses, or hot moist sponges, have been found to afford relief.

ŒDEMA OF THE LARYNX

Symptoms.—Clinically two forms may be distinguished: the acute, where symptoms are severe and of rapid onset; and the chronic, where œdema slowly develops without any special symptoms. The symptoms of the acute form are dyspnœa, local pain or discomfort, dysphagia, and alteration or loss of voice. According to the situation of the swelling, inspiration or expiration may be chiefly affected, inspiration being difficult and perhaps

attended with stridor if the aryteno-epiglottidean folds be involved, while the expiration is laboured if the swelling be seated in the ventricular bands and the posterior aspect of the epiglottis. The patient feels as if there were a foreign body in the throat. He has difficulty or pain in swallowing, and is apt to choke when he attempts to do so, and he hawks up saliva which accumulates in the pyriform sinus. The voice is thick and muffled, or there may be aphonia. On laryngoscopic examination there is seen to be swelling of the epiglottis and aryteno-epiglottidean folds. The mucous membrane is usually pale, except at the borders of the swelling, where congestion may be present. The epiglottis is usually erect and tense, standing out like a roll and often preventing a view of the rest of the larynx. The aryteno-epiglottidean folds form plum-shaped bodies, which may almost meet in the middle line. In subglottic œdema two red fleshy swellings may be seen below the cords. The prognosis is most unfavourable in septic cases. General œdema is more serious than the unilateral form. The outlook is improved by early recognition of the condition, and by the adoption of the appropriate treatment.

Treatment.—The general treatment is the same as that of acute laryngitis, but it is specially important that ice should be given to suck, and that food should consist of liquids or soft solids; and if even these be difficult to swallow, rectal feeding should be employed. Leeches may be applied to the sides of the larynx. Subcutaneous injections of pilocarpine (gr. $\frac{1}{8}$), and in the œdema of iodism, bicarbonate of soda, internally, have sometimes proved useful. Prompt local treatment is necessary in cases where there are urgent symptoms. To reduce the local œdema, the larynx, after swabbing with 20 per cent solution of cocaine, should be scarified by means of the guarded laryngeal lancet or a long, sharp-pointed bistoury protected nearly to its point by strips of adhesive plaster. This should be followed by steam inhalations and gargling with warm water. Tracheotomy should be performed if dyspnoea be urgent and be not soon relieved by other measures.

CHRONIC LARYNGITIS

Symptoms.—The most characteristic symptom is alteration of the voice, which usually takes the form of hoarseness. Hoarseness is most marked in the morning and on using the voice after rest, especially when making some special effort such as in singing or preaching. Sometimes there is complete aphonia, and sometimes

the compass, quality, and power of the singing voice are much impaired. A feeling of dryness, burning, or pricking in the throat is often combined with a desire to clear it, and a little whitish-gray viscid secretion may be coughed up. The laryngoscopic appearances are redness and thickening of the cords, with possibly some adherent secretion, and injection of the vessels of the epiglottis. In *laryngitis sicca* dry crusts of inspissated secretion are coughed up with difficulty. The crusts may be blood-stained, or in some cases pure blood may be brought up. The presence of large crusts on the ventricular bands or cords may give rise to attacks of dyspnoea. The crusts may be visible on laryngoscopic examination, especially near the posterior commissure. Two other special forms of chronic laryngitis merit separate mention, but their symptoms do not essentially differ from those of the ordinary affection. *Pachydermia laryngis* is a condition in which, as described at p. 61, there are symmetrical fleshy-looking thickenings of the cords, usually occupying the processus vocales but sometimes the anterior thirds, it being characteristic that there is on one of the swellings a cup-shaped depression, and that the mobility of the cords is not interfered with. *Singers' nodes* are of a similar nature, the local thickening being in the form of a small round nodule seated on the upper surface and free border of one or both cords, generally at the junction of the anterior with the middle thirds.

Diagnosis.—It is important to distinguish laryngeal tuberculosis from ordinary chronic laryngitis. The sputum, if any can be obtained, should be examined for tubercle bacilli, and the symptoms and physical signs of pulmonary tuberculosis should be carefully looked for. In pachydermia laryngis and singers' nodes the appearances are in themselves characteristic. Impaired mobility, thickening, and congestion limited to one cord should excite a suspicion of malignant disease, especially in persons past middle life.

Prognosis and treatment.—The prognosis is good provided the cause be removed and appropriate treatment adopted. It is of prime importance to secure complete rest to the voice, abstinence from tobacco and alcohol, and purity of the atmosphere which the patient breathes. Before starting local treatment, the nasal passages should be carefully examined, and any local condition interfering with free nasal respiration remedied. Astringent local applications to the vocal cords are of great value. Solution of chloride of zinc, 20 grs. to the ounce, should be applied by means of a laryngeal brush, daily at first and afterwards less frequently. In severe forms stronger solutions of chloride of zinc (60 grs. to

the ounce), or solution of nitrate of silver (20 to 80 grs. to the ounce) may be employed. Astringent sprays, such as chloride of zinc (2 grs. to the ounce) and perchloride of iron (3 grs. to the ounce), are useful as the malady is passing off. In pachydermia laryngis, Lugol's solution (iodine 2, potass. iod. 3, water 40 parts), dilute acetic acid (3 per cent), or a saturated alcoholic solution of salicylic acid may be locally applied, while iodide of potassium is given internally. In any form of chronic laryngitis, after local treatment, change of air is most important, and a course of treatment at Ems or Aix-les-Bains often proves very beneficial.

PERICHONDritis OF THE LARYNX

Symptoms.—In most of the secondary cases the symptoms are not characteristic, being chiefly those of the primary affection.

Exceptionally there is an acute invasion, with chilliness or an actual rigor and rise of temperature. There may be a dull aching referred to the larynx, with tenderness on pressure; and if the posterior surface of the cricoid be involved, there are pain and difficulty in swallowing. Alteration of the voice and dyspnea are usual when the glottis is encroached on. On laryngoscopic examination a smooth or nodular unilateral swelling may be seen, with not uncommonly immobility of the corresponding vocal cord.

When the cricoid is affected, the situation of the swelling is either subglottic or interarytenoid, or on the posterior surface. If, as is usual, necrosis occur, crepitus may be felt on pressing the larynx. When an abscess has discharged, necrosed cartilage may be recognised by means of the laryngeal probe.

The **diagnosis** is often extremely difficult, especially in connection with malignant disease and syphilis. The usual limitation to one cartilage, and, after the discharge of the abscess, the less angry look of the swelling, are important points in the diagnosis from malignant disease.

The **prognosis**, as regards recovery of voice and freedom of breathing, is generally most unfavourable. Tracheotomy is often necessary on account of the degree of stenosis.

The **treatment** is similar to that recommended for œdema. The strength should be maintained by careful feeding, and after the acute stage is over the necrosed sequestrum removed when within reach. When the condition is due to a foreign body, thyrotomy should be performed. In syphilitic cases iodide of potassium must be freely given.

DISEASE OF THE CRICO-ARYTENOID JOINT

The joint may be completely fixed or ankylosed in any position, but in true ankylosis the position is such that the cord lies somewhere between the phonatory and cadaveric positions. If ankylosis be complete, the cord is motionless; if incomplete, its movements are impaired or jerky. If there be luxation of the joint, the arytenoid occupies an abnormal situation, and the contour of the larynx is distorted. In addition to impairment of mobility, there is usually tumefaction over the arytenoid, and this may be considerable. The chief symptom is alteration of the voice. Dyspnoea is marked in the case when both cords are fixed near the middle line.

Fixity of the cord from ankylosis may be difficult to distinguish from fixity due to paralysis of the recurrent laryngeal, and thus may give rise to a suspicion of aneurysm. The presence of tumefaction, and an abnormal position of the arytenoid, point to ankylosis. The condition requires treatment only when dyspnoea is urgent, in which case those measures may be employed which are useful in stenosis from other causes.

LARYNGEAL STENOSIS

The principal symptom of stenosis, from whatever cause it arises, is dyspnoea. When the stenosis is acute it should be relieved by either tracheotomy or intubation. The same means may be employed in chronic cases, but tracheotomy is to be preferred when the obstruction is permanent, for with a tube in the larynx the patient can only speak in whispers, while with a tube in the trachea he can, by closing the opening, speak in an audible tone. Dilatation of the stenosed opening may be attempted by means of Schrötter's bougies or by the successive introduction of increasingly large O'Dwyer's tubes.

SYPHILIS OF THE LARYNX

The larynx is affected in from 3 to 4 per cent of all cases of syphilis, but seriously so in a much smaller proportion.

The **symptoms** are not very characteristic, and often nothing beyond hoarseness or aphonia is observed. Both in secondary and in tertiary syphilis the voice may have a peculiarly raucous character. Cough is rarely troublesome. Persistent pain, it is

important to note, is generally absent save in the case of the graver forms of lesion. Dysphagia is seldom marked except when the epiglottis or the posterior surface of the cricoid cartilage is affected, in which case pain, as well as difficulty in swallowing, may be present. If the glottic opening be narrowed, dyspnoea will likely be present. The laryngoscopic appearances may be gathered from the description already given of the morbid changes.

The **diagnosis** usually depends on the history or on the presence elsewhere of syphilitic lesions or their traces. In cases where the history is negative, and other evidence of syphilis is wanting, there may be considerable difficulty in distinguishing laryngeal syphilis from tubercle or new growth. Syphilitic ulceration is more rapidly progressive, extends more deeply, is less frequently multiple, and is more generally free from pain than tuberculous ulceration. Doubt must often exist in the case of new growth, especially the infiltrating form of carcinoma, and can only be removed by a thorough trial of antisyphilitic treatment. Glandular infiltration points to malignant disease, while absence of pain and rapid extension of ulceration suggest syphilis. If an ulcer do not show signs of healing, or if a growth do not diminish under the administration of iodide of potassium, it is not syphilitic.

The **prognosis** as regards life is good when specific treatment is carried out. Ulceration, however, leaves behind it scars, from the contraction of which serious deformity may arise. The voice may be permanently hoarse, or stenosis may lead to dyspnoea. The supervention of acute oedema or perichondritis is always serious.

Treatment must be mainly constitutional, and does not differ from that of other forms of syphilis. It should, however, be vigorous in the presence of ulceration, oedema, or general infiltration. Where a rapid effect is desired, mercurial inunction may be employed, or 6 to 9 minims of the gray oil (one part of mercurial ointment in two parts of sterilised oil) may be daily injected into the deeper subcutaneous tissue or muscle of the buttock or back. In the later lesions iodide of potassium should be given in doses of 10 to 20 grains three times a day.

TUBERCULOSIS OF THE LARYNX

Symptoms.—The symptoms at first do not differ from those of chronic laryngitis. Hoarseness is usually an early symptom, while later in the disease the voice may be lost altogether. Dysphagia is common in severe cases. Swallowing is often painful, or is

attended with difficulty on account of the obstruction caused by the swelling, or on account of food entering the larynx from imperfect closure. There is sometimes a constant feeling of soreness or rawness referred to the larynx. Cough is seldom absent, and in the later stages may be severe and paroxysmal. The other symptoms usually present are those of chronic pulmonary tuberculosis. As a rule, the general condition is bad and the patient has a decidedly cachectic look, when the disease is at all extensive.

The **laryngoscopic appearances** may be deduced from the description of the morbid anatomy already given. Pallor of the mucous membrane is an early sign often, but not constantly, present. The most characteristic of the lesions, as seen by the laryngoscope, are projecting granulations in the interarytenoid fold, ulceration at the processus vocalis, a split or terraced appearance of the cords, pear-shaped swelling of the aryteno-epiglottidean folds, and a turban or sausage-shaped swelling of the epiglottis.

Diagnosis.—The nature of the affection will usually be correctly diagnosed from the presence of lesions, such as have been described, together with evidence of tubercle in the lungs or elsewhere, or the discovery of tubercle bacilli in the expectoration. Persistent chronic laryngitis should always be regarded with suspicion, especially when limited to one cord. Reference has already been made to the diagnosis from syphilis under the heading of laryngeal syphilis. The appearances of laryngeal tubercle may also closely resemble those of malignant disease; but in the former, tumour formation is, as a rule, less definite, and lesions are less frequently limited to one side, than in the latter. Patients under forty are more liable to laryngeal tuberculosis, those over forty to malignant growth. It must be borne in mind, with regard to both syphilis and malignant disease, that they may occur in tuberculous subjects, and may be combined with tubercle in the larynx itself.

Prognosis.—The prognosis of laryngeal tuberculosis is always grave, but the less severe cases in which pulmonary tuberculosis is arrested or quiescent sometimes do extremely well under appropriate constitutional and local treatment. Little hope of improvement can be held out when the lungs are extensively diseased and the general condition is bad. Severe and continued pain, difficulty of swallowing, and great irritability of the pharynx and larynx are most unfavourable signs.

Treatment.—It must in the first place be remembered that there is always need for general constitutional treatment. This should be carried out on the same lines as in the case of chronic

pulmonary tuberculosis, which is practically always present when the larynx is extensively involved. Purity of the air and absence of dust are most important elements. Sanatorium treatment on open-air lines is sometimes attended with surprisingly good results, but, as a rule, a very dry air, such as that in the high altitude resorts, is not well borne. Prolonged rest to the larynx is of great value, and even whispering must be forbidden in some cases. It is almost unnecessary to say that smoking cannot be permitted. Coughing, when ineffectual in removing secretion, is harmful, and should be restrained or checked.

Local treatment may be either symptomatic or applied with the object of curing the disease.

When there is pain in swallowing, cocaine pastilles or lozenges or a cocaine spray (2 to 5 per cent) may be used before food is taken. Soft or semi-solid food is more easily swallowed than solid or liquid food. Liquid food often sets up coughing, and is more likely to do so when sipped than when taken in a long draught. Liquid food may with advantage, in such cases, be thickened with isinglass, arrowroot, or corn-flour. Sometimes it will be found that the patient can swallow better in the prone position, or with the head hanging down, as when drinking out of a stream, than in the ordinary position. When an adequate amount of nourishment cannot be taken, the patient should be fed by means of a soft œsophageal tube, or by nutritive enemata.

Soothing inhalations are useful for the relief of soreness and cough. These may be moist, such as vapor benzoini, or dry, such as menthol or chloroform inhalation (Brompton Hosp. Pharmac.).

Insufflations of finely powdered iodoform (gr. i.), orthoform (gr. ii.), cocaine (gr. $\frac{1}{4}$ to $\frac{1}{2}$), or morphine (gr. $\frac{1}{8}$ to $\frac{1}{2}$), diluted with starch, are helpful in relieving pain and in producing a more healthy condition of the ulcerated surfaces. Antiseptics, such as solutions of carbolic acid or menthol, are sometimes applied with the laryngeal brush with benefit.

The most successful local treatment which has been employed is the application of lactic acid, but it is only beneficial in certain cases. It should not be employed before there is actual ulceration, or when there is extensive ulceration or infiltration, much œdema or perichondritis, marked irritability of the larynx, or active and advanced lung disease.

After a thorough preliminary cocainisation of the larynx, lactic acid is applied on cotton-wool attached to a laryngeal screw-holder,

and is well rubbed into the ulcerated surface. To begin with a 50 per cent solution may be employed, but the strength should soon be increased until the pure acid is used. If the pure acid be used and the application be thorough, a week or a fortnight may elapse before it is repeated. Only a few applications altogether should be made.

More radical measures, such as cutting or curetting, have been employed and sometimes combined with lactic acid treatment with the object of removing diseased tissue or of preparing a surface for the application of the acid. It is not generally considered advisable to employ the curette or cutting forceps in the absence of ulceration, and the cases where these methods are likely to benefit at all are not numerous. Their use, it is needless to say, by any but skilful and experienced surgeons cannot be advised.

LARYNGEAL GROWTHS

Symptoms.—*Benign growths.*—The most common and often the only symptom is alteration of voice, which may be hoarse, croaking, harsh, or aphonic, according to the position and extent of the growth. Small growths often more seriously affect the voice than large. Dyspnœa, sometimes paroxysmal, is present if the opening of the glottis be encroached on. It is increased by exertion and by the presence of catarrh; cough is occasionally troublesome, and in the case of papillomata in children has often a croupy character. Pain is usually absent, and there is no dysphagia unless the growth be situated on the upper surface of the epiglottis. The laryngoscopic appearances may be gathered from the account given of the morbid anatomy of the growths.

Malignant growths.—In many cases alteration of voice is usually the earliest symptom, as in the case of benign growths, and may precede other symptoms by months. Hoarseness, caused as much by impaired mobility of the cord as by the actual growth itself, after a time gives place to aphonia. The voice may remain normal in cases where the posterior surface of the cricoid, the epiglottis, or the aryteno-epiglottidean fold is the seat of the growth.

Pain, sometimes radiating to the ear, and tenderness on the affected side are occasionally present. Dysphagia occurs in the case of growths of the epiglottis or the posterior surface of the larynx, and is not uncommonly associated with salivation and increased secretion from the mucous glands. In cases of ulcerating growth there is a foetid secretion which makes the breath offensive, and hæmorrhages

may occur or the secretion may be blood-stained. Dyspnœa is usually a late symptom, but then may be severe.

Cancerous cachexia is seldom marked except in cases where there has been extension to neighbouring parts. Enlargement of glands, the posterior cervical and those beneath the sternomastoid, occurs in cases where the growth has extended to the pharynx.

The **laryngoscopic appearances** may be deduced from the morbid anatomy. In some cases at first there is only diffuse hyperæmia of one cord, a condition which should always excite a suspicion that the case is not one of simple chronic laryngitis. In other cases there are warty or infiltrating growths on one cord, often situated at its middle, and it is important to note that the mobility of the cord is impaired from an early period. The growth is generally surrounded by a circumscribed pinkish zone of congestion, which contrasts with the whiteness of the remainder of the cord. Although sometimes the tumour changes little in appearance, more commonly it grows rapidly, spreads to neighbouring parts, and ulcerates, perichondritis and exfoliation of cartilage occurring as secondary processes.

Diagnosis.—In the case of benign growths the chief difficulty is to distinguish them from the malignant. Here the age of the patient and the appearances will help. The malignant growth is more intimately blended with the tissues, it early ulcerates and tends to bleed, and it rapidly recurs after removal. When the cord is the seat of malignant growth, its mobility is usually greatly interfered with. It may be noted that if we except pachydermia the posterior thirds of the cords are scarcely ever the seat of benign growths. In doubtful cases a portion should be removed for microscopical examination, but not unless the patient be willing to undergo radical treatment in the event of the growth being shown to be malignant. The diagnosis from chronic laryngitis has been already referred to. The clinical points which have been mentioned as aids in the differential diagnosis of syphilis and tubercle from malignant disease may here be conveniently recapitulated in tabular form.

SYPHILIS.	TUBERCLE.	MALIGNANT DISEASE.
Usual age twenty-five to fifty; history of syphilis or evidences of other syphilitic affections.	Usual age twenty to forty; evidences of pulmonary tubercle usually present.	Usual age forty to sixty; not commonly combined with pulmonary tubercle.
Little irritability of pharynx.	Marked irritability and anæmia of pharynx.	Definite tumour formation usually surrounded by intensely congested zone.
Tumour, if present, usually a smooth red tumefaction.	Tumour formation seldom definite.	Generally at first limited to one side.
Not commonly unilateral.	Seldom limited to one side except when cord is affected.	Mobility of cord early affected.
Mobility of cords not affected until late period from perichondritis or ankylosis.	Mobility unaffected.	Ulceration usually combined with tumour; marked tendency to bleed.
Ulceration rapidly progressive, large, deep, sometimes "crater-like," surrounded by considerable area of inflammatory swelling, but not commonly multiple.	Ulceration usually preceded by pale cedematous-looking infiltration, slowly progressive, rather superficial than deep, worm-eaten appearance, being often multiple.	Pain common.
Pain much less common than in tubercle and cancer.	Pain common.	Glandular infiltration, when present, an important sign.
Glandular infiltration rare, but, if present, little marked.	Glandular infiltration absent.	No local change from iodide of potassium.
Healing of ulcer and diminution of tumour when iodide of potassium is given.	No local change from iodide of potassium.	

Prognosis.—In benign growths the prognosis is on the whole good as regards life and health, and the voice can usually be restored when radical treatment is skilfully carried out. An exception must be made, however, in the case of multiple papillomata in children, when thyrotomy has been performed, or when, to prevent suffocation, tracheotomy has been necessary. It has been alleged that removal of benign growths by intralaryngeal operation is sometimes followed by malignant disease at a later period, but statistical evidence shows that this event is very rare, and more likely to occur in cases which are left to themselves than in those operated on.

In malignant growth the prognosis is most unfavourable in old people, and in cases where the growth is extensive. When the

growth is limited and the patient in middle life, radical operation offers a fair chance of cure.

Treatment.—Whenever possible, as in the great majority of cases it is, benign growths should be removed intralaryngeally by means of forceps or snares. Small sessile fibromata, giving rise to little inconvenience and difficult to remove without damage to the cord, may be left. In the case of young children, in whom it is seldom possible to employ intralaryngeal methods with success, the removal of the growths should be postponed, but tracheotomy is advisable if dyspnœa be present. In certain exceptional cases thyrotomy or subhyoid pharyngotomy may be necessary for the removal of the growths, and in the case of malignant growths one of these operations, followed by excision of the tumour and partial laryngectomy, offers the best chance; but it is only in intrinsic and limited cases that operation is likely to be crowned with success. In inoperable malignant cases life may be prolonged and comfort promoted by judicious feeding, and tracheotomy should be performed when the breathing is difficult.

LARYNGEAL NEUROSES

SPASMODIC AFFECTIONS.—Spasm of the glottis in children, or laryngismus stridulus, an affection which has received a variety of other names, such as spasmodic croup, cerebral croup, child-crowing, thymic asthma, Millar's asthma, and angina spastica, has already been described in Vol. III. p. 314.

Spasm of the glottis in adults is usually reflex, resulting from irritation of the larynx by foreign bodies, new growths, an elongated uvula, etc., or from irritation of the vagus or of both recurrent laryngeal nerves by aneurysms or mediastinal growths. In other cases it is the result of some disease of the central nervous system, such as locomotor ataxy, tetany, hysteria, or hydrophobia.

The **symptoms** are similar to those of laryngismus, but are usually less severe, and in many cases nothing more is noticeable than that several succeeding inspirations are stridulous. Rarely the spasm is prolonged until there is loss of consciousness or death occurs.

The inhalation of chloroform and the use of a 2 per cent cocaine spray twice daily have proved beneficial.

NEUROSES OF CO-ORDINATION.—Irregular movements of the vocal cords may be observed in chorea and disseminated sclerosis. Functional inspiratory spasm occurs in cases of hysteria, and pro-

duces symptoms similar to those of bilateral paralysis of the abductors. It can be distinguished from the latter by examination with the laryngoscope, when the cords may be observed occasionally to separate well during expiration.

NERVOUS LARYNGEAL COUGH—BARKING COUGH OF PUBERTY.—Either sex may be the subject of this troublesome affection, but it is chiefly met with in young people between the ages of sixteen and twenty. It is characterised by a peculiar harsh cough, usually single and abrupt, and frequently repeated at short intervals. The cough is often extremely noisy, and the noise, which is sometimes shrill and metallic, sometimes deep and vibratory, has been variously compared to the barking of a dog, the quacking of a duck, or the deep tones of a clarionet played fortissimo. The cough may be very troublesome at night, but usually ceases during sleep. As a rule the general health remains good, there is no expectoration and no alteration of the voice, and the laryngoscopic appearances are normal.

The affection may last for weeks, months, or even years.

The most successful remedy has been found to be a sea-voyage. Other forms of treatment have not on the whole proved of much avail, but of these the most useful have been the local application of cocaine and the administration of sulphate of iron and bromide of potassium internally.

PHONIC SPASM—DYSPHONIA SPASTICA—SPASM OF THE TENSORS OF THE VOCAL CORDS.—This affection is a form of spasm of the tensors and adductors which comes on only on attempted phonation. It occurs among adults, and chiefly in men, especially among those who have to use their voices professionally. At first the patient, after giving utterance to a few words in a natural tone, is unable to proceed, the voice becoming weak and strained, or disappearing altogether. Later, any attempt at phonation produces closure of the glottis, and all efforts to force the air through the closed opening prove futile. On laryngoscopic examination, the cords are seen to act normally during respiration, but on attempted phonation they come forcibly together and may overlap. As soon as the effort to speak is abandoned, the glottis opens. Whispering is usually unattended with difficulty, but sometimes even that induces spasm. Treatment is usually ineffectual. Breathing and elocutionary exercises, such as are recommended for stammering, give the best results.

LARYNGEAL VERTIGO is the name which has been given to a rare form of vertigo which appears to be connected with spasm of

the larynx. A tickling sensation in the larynx is followed by cough, giddiness, and brief unconsciousness. Attacks occur at intervals, varying from a few days to several months. Change of air and bromide of potassium sometimes benefit these cases.

PARALYSIS OF THE LARYNGEAL MUSCLES has been already discussed under the heading of the Vagus Nerve, Vol. III. p. 245, and we shall only here consider two forms which give rise to characteristic symptoms.

PARALYSIS OF THE ADDUCTORS.—*Functional aphonia*.—Paralysis of the adductors of the larynx is almost invariably bilateral, and is a functional affection independent of any organic disease. It is principally met with in the case of young women, but may occur at any age and in either sex. It sometimes follows catarrhal laryngitis, or arises in the course of phthisis, anæmia, or other conditions of debility, but its causes are those of hysteria in general.

Symptoms.—The usual symptom is loss of voice, which may be constant or occasional, but commonly the former. In some cases the voice is not lost, but there is hoarseness. The involuntary sounds of cough and sneeze are preserved, an important point of distinction from aphonia due to local organic disease. The affection comes on more or less suddenly. On laryngoscopic examination the larynx is seen to be normal, but on attempted phonation the cords do not meet in the middle line. There is usually diminished sensitiveness of the pharynx, which facilitates examination. The diagnosis presents no difficulties after a laryngoscopic examination has been made.

Treatment.—It is of great importance to attend to the general health and to remedy any associated condition, such as anæmia. Strychnine by the mouth or preferably by subcutaneous injection appears to benefit in some cases, and shower-baths may be given with advantage. Sometimes the voice is brought back simply as the result of the examination with the laryngoscope or the application of a laryngeal brush to the larynx; but commonly to produce an immediate restoration of the voice it is necessary to employ the induced current. It is important to know, and this can be said from a large experience of these cases, that it is usually immaterial to what part of the body the current is applied. The essential point is to use a strong current and apply it suddenly, so as to produce a powerful impression. The laryngeal electrode, by means of which the current is applied directly to the larynx, is convenient for use, but is not necessary for cure. The application of the strong induced current usually causes the patient

to cry out, after which she should be encouraged to count and repeat sentences slowly in a natural voice. A second application of the current may be necessary if the voice be still not quite normal. It must be borne in mind that there is a tendency to relapse, and the patient should be kept under observation for some time. The patient must exercise the voice regularly by reading and counting aloud.

PARALYSIS OF THE ABDUCTORS.—In bilateral paralysis the cords remain near the middle line, both in inspiration and expiration. An important symptom is stridulous inspiration, which, although absent when the patient is at rest, is readily induced by exertion or excitement, and is generally present during sleep. The sound so produced may disturb a whole household at night. Paroxysmal attacks of dyspnoea are common.

Unilateral paralysis often gives rise to no symptoms, and at the most there is slight hoarseness.

Treatment of these affections must be directed to their cause. In the case of bilateral paralysis, tracheotomy is advisable if dyspnoea be a marked symptom.

CONGENITAL STRIDOR OF INFANTS.—The causation of this affection is uncertain, although to explain it a good many theories have been put forward. It has been attributed to paralysis of the abductors, an enlarged thymus, a congenital malformation of the upper aperture of the larynx, and to an ill co-ordinated spasmodic action of the muscles of respiration.

Shortly after birth the infant, normal otherwise, is observed to breathe in a peculiar noisy fashion. Inspiration is attended with a sound which is usually croaking, but becomes high-pitched and crowing when long or deep breaths are taken. Expiration is usually noiseless, but occasionally is accompanied with a short croak. With the exception of a few brief intervals when the breathing is quiet, the stridor is constant while the child is awake, and is apt to be intensified by emotional excitement, the exertion of sucking, or exposure to a draught of cold air, and it sometimes persists during sleep. There is marked inspiratory retraction of the lower part of the chest. Apart from the peculiar noise of the breathing, the child appears well; its cough and cry are normal, and there is neither distress in breathing nor cyanosis. During the first few months of life the stridor increases in loudness. It then remains stationary for a few months, after which it generally lessens, disappearing altogether during the second year.

HECTOR MACKENZIE.

DISEASES OF THE TRACHEA AND BRONCHI

ACUTE TRACHEITIS AND BRONCHITIS

The morbid condition denoted by these terms is an inflammation of the mucous membrane of the air-passages of the lower respiratory tract, the anatomical changes and the pathology of which have been considered at p. 61.

Symptoms.—In the mildest form the inflammation, which usually starts in the upper respiratory tract, does not extend farther than the trachea (acute catarrhal tracheitis). The symptoms may be limited to a sense of soreness referred to the region of the trachea, and cough, which is hacking, violent, or paroxysmal, and probably accompanied by little expectoration. Tracheitis is a frequent complication of influenza, but in the ordinary form there is little constitutional disturbance.

When the larger bronchi as well as the trachea are involved (acute tracheitis and bronchitis) the illness is more severe, and the main symptoms are cough with expectoration, and dyspnoea, with or without pyrexia. The illness may come on either gradually or suddenly. A gradual mode of onset is the more common, and then the illness usually begins with catarrh of the upper air-passages, starting in different cases in the nose, nasopharynx, or fauces, or else in the larynx with consequent hoarseness or aphonia. Sometimes, however, from the beginning the trachea and bronchi are affected, and the earliest symptoms are soreness, oppression, or tightness in the chest, with cough and expectoration. The catarrhal symptoms are usually accompanied with general malaise.

The sudden or rarer mode of onset is most frequent in elderly subjects, but sometimes occurs also in children or adolescents. Dyspnoea, with or without cough, sets in acutely, the illness in some cases closely resembling spasmodic asthma, from which for a time it may be quite impossible to distinguish it.

The cough, which at first is usually dry, and either short, hacking, and frequent, or more or less severely paroxysmal, is attended with tickling sensations in the throat and soreness or burning in the chest behind the sternum. The straining produced by the cough sometimes gives rise to severe pains in one or both sides. Later, expectoration occurs which, to begin with, is scant

and consists of thick and viscid mucus, but after two or three days becomes more abundant, thin, and glairy like white of egg, and frothy from the presence of air. The thin secretion contains few cells, and these for the most part columnar epithelial cells which have lost their cilia; but there are opaque streaks, at first whitish, then greenish, which consist chiefly of leucocytes, and occasionally there are traces of blood.

The quantity expectorated in this stage varies in different cases from 1 or 2 to 10 or 20 ounces in the twenty-four hours. The thin, glairy, mucous sputum (*sputum crudum*) gradually changes in character, becoming muco-purulent, yellowish or greenish-gray, thick, and opaque (*sputum coctum*). Usually when the sputa become opaque, expectoration becomes easier, and pyrexia, if present, subsides. With the occurrence of exacerbations there may be a return to the mucoid form. The more opaque sputa are usually partially confluent and sometimes nummulated, but in some cases of a severe type in elderly people the sputa are puriform and diffuent.

Dyspnoea is an almost constant symptom, but differs greatly in degree in individual cases. It is as a rule more intense in children than in adults, and in them the respiration rate may be from 40 to 80 in the minute. In mild cases in adults the respiration may be only slightly hurried, while in severe cases the rate may be 40 or even 50 to the minute. The rapidity of the breathing may be paroxysmal, and increased by excitement, cough, or exertion. In some cases the dyspnoea assumes an asthmatic character. There is often more expiratory than inspiratory difficulty, especially when the smaller tubes are affected, and in children an apparent pause may be observed at the beginning of expiration instead of at the end. Orthopnoea usually occurs in severe cases, and in the more intense cases the breathing assumes the Cheyne Stokes type.

Cyanosis may be observed in cases of some severity. The face is either pale and livid or flushed and dusky, while the lips and hands show varying degrees of blueness.

Pyrexia is more constantly present in children than in adults, among whom more than half the cases run an afebrile course. In adults a normal temperature in the morning and a slight rise in the evening is the usual type of pyrexia, and even in children the temperature is rarely high unless broncho-pneumonia co-exist. In exceptional cases, both in children and adults, a remittent type of fever occurs, the morning temperatures being 101° or 102° , and the evening a degree or two higher, the fever generally declining by lysis before the end of the second week.

The rate of the pulse is generally an index of the severity of the illness. In slight cases the pulse is little if at all quickened, while in serious attacks the rapidity may be out of all proportion to the temperature. In the very young and the old the pulse may be very rapid, and in the latter it is frequently irregular.

The skin, except just at the onset, generally acts freely, and in severe cases profuse perspirations are the rule, especially after paroxysms of coughing.

Probably as the result of catarrh of the mucous membrane, there are usually signs of gastro-intestinal disturbance, such as anorexia, furring of the tongue, and constipation, or, in the case of children, diarrhoea, while vomiting not infrequently follows a severe paroxysm of coughing. When there is fever, the urine is usually scanty, high-coloured, and loaded with lithates, becoming paler and more abundant as the illness declines. Affections of the nervous system are usually limited to headache and pains in the limbs, but in old people and in children there may be nocturnal delirium.

Physical signs.—In the milder form of the disease there are few abnormal physical signs, the only adventitious sounds being sonorous rhonchi, audible on auscultation in front or behind. In cases of a more serious type, the chest is fully expanded and the respiratory movements are exaggerated, those of the chest-wall being principally up and down. In the case of children, retraction of the lower end of the sternum and lower ribs occurs during inspiration when there is a high degree of dyspnoea. On palpation rhonchal fremitus is sometimes to be felt, and the vocal fremitus is increased. The percussion note is normal unless emphysema be present, when it is drum-like and over-resonant, and the resonant area exceeds the normal limits. Dulness may be present at the bases from collapse or broncho-pneumonia. On auscultation the breath sounds, especially in the upper part of the chest, are harsh and exaggerated, expiration being relatively prolonged. Suppression of respiratory sounds from obstruction of the bronchi may be temporarily observed in some situations. Adventitious sounds chiefly consist of sibilant and sonorous rhonchi, depending on the presence of mucus or inflammatory swelling of the tubes. After cough and the removal or displacement of mucus, the rhonchi may temporarily disappear. In the stage when the secretion is thin, crepitations are audible, and are usually generally distributed. When crepitations are limited to the base they probably depend on the presence of broncho-pneumonia or œdema.

Course and duration.—Individual cases vary greatly in their severity, progress, and duration. The milder forms are attended with but little constitutional disturbance, and convalescence may set in after a few days' illness, but even mild cases may be protracted in duration. Cases complicated with emphysema and cases occurring at the two extremes of life are always serious, and grave symptoms may rapidly set in. The average duration is about three weeks, and it is the exception for the illness to last less than two weeks. Relapses, which are of common occurrence, may come on at any stage, even during convalescence, and are particularly to be feared in the case of elderly subjects.

Diagnosis.—It is only seldom that any difficulty of diagnosis is likely to arise. In the early stages it may be impossible to exclude whooping-cough and measles, which are likely or not, according as there is or is not a history of contagion. Bronchitis is sometimes so prominent a feature of typhoid fever that the primary disease may be overlooked. In such a case, pyrexia, prostration, and headache are marked symptoms, and although there are abundant rhonchi there is probably but little cough or expectoration. Enlargement of the spleen and the presence of spots should be looked for, and the blood should be tested for the Widal reaction. Pneumonia may resemble bronchitis, but the more sudden onset, the higher temperature, the pleuritic stitch, and the altered pulse-respiration ratio will usually serve to distinguish the one from the other. The pulmonary congestion of chronic cardiac disease, especially mitral stenosis, may also sometimes closely resemble acute bronchitis and be mistaken for it if the heart affection be not discovered. The greatest difficulty of diagnosis, perhaps, arises in recognising the co-existence of acute pulmonary tuberculosis. The occurrence of high and persistent fever of a slightly remittent or inverse type, marked prostration, early delirium, hæmoptysis, and the limitation of râles to the upper lobe of one lung are all in favour of associated tubercle.

Prognosis.—Except in early life and old age a fatal result is uncommon, even with the existence of emphysema, when the disease is primary, but it must be remembered that acute bronchitis is a common termination of many chronic maladies. Influenza, measles, whooping-cough, tubercle, heart disease, arterial degeneration, kidney disease, and rickets add to the danger. The most serious symptoms are cyanosis, extreme rapidity or irregularity of the respiration, extreme dyspnoea, and prostration, together with delirium or coma in old people or convulsions in children. It is a bad sign

when the secretion becomes thicker and more scanty without corresponding improvement in the general signs. It should be borne in mind that one attack of acute bronchitis predisposes to another.

Treatment.—The patient should be kept in bed in a well-ventilated room, the temperature of which is maintained at about 62° F.

At the outset a hot bath or such a modified form of Turkish bath as the patient can have at home may be given with advantage. When there is difficulty of breathing and little or no secretion, as in the early stage, moisture in the atmosphere is beneficial, and this can be attained to a certain extent by the use of a "bronchitis kettle." Steam inhalations are also useful. Later, when secretion is free, steam is neither necessary nor beneficial. Linseed or linseed and mustard poultices or turpentine stupes afford great relief when there is tightness and soreness of the chest.

In the early stage, diaphoretic and saline remedies, combined, except in the case of young children, with small doses of opium, are most useful. A few five-grain doses of Dover's powder may be given at first, or a mixture such as the following may be prescribed :—

℞ Vin. ipecac. ʒj.
Tr. camph. co. ʒij.
Sp. æth. nitros.
Extr. glyc. liq. āā ʒiij.
Liq. ammon. acet. ʒiss.
Aquam ad ʒvj.

Ft. mist. Sig.—Two tablespoonfuls to be taken three times a day.

Some recommend at the beginning small doses of aconite (one minim of the tincture) every half-hour until free perspiration occurs. For the violent paroxysmal cough of tracheitis such a linctus as the following may be given :—

℞ Morphin. hydrochlor. gr. ss.
Apomorphin. hydrochlor. gr. $\frac{3}{4}$.
Acid. hydrochlor. dil. mxx.
Syr. prun. Virgin. ʒss.
Aquam ad ʒij.

Ft. linct. Dose ʒj. occasionally.

For the same condition heroin hydrochloride in doses of $\frac{1}{12}$ grain is useful.

The bowels should be kept open, and for this purpose a saline aperient is most suitable. The diet should be plain and easily

digested, during the acute stage consisting largely of milk, eggs, rusks, and beef-tea. Warm drinks, such as hot milk diluted with Ems water, help to relieve cough, and may be freely given; but no alcohol should be allowed at this stage unless the patient be habituated to its use.

When expectoration is free, carbonate of ammonia, squill, and senega may be given with advantage, and ipecacuanha is as suitable at this period as it is in the early stage. The following mixture may be substituted for the first:—

℞ Ammon. carb. gr. xxx.
 Vin. ipecac. ℥j.
 Tr. scill. ℥iss.
 Sp. chlor. ℥ss.
 Infus. seneg. ad ℥vj.

Ft. mist. Sig.—Two tablespoonfuls to be taken three times a day.

In some cases where the dyspnœa appears to be due to spasm, iodide of potassium, stramonium, and lobelia prove as useful as in ordinary asthma. In the case of children, when there is much dyspnœa, the result of the accumulation of mucus in the tubes, emetics are indicated, ipecacuanha wine in drachm doses being repeated until vomiting occurs.

In severe attacks of a sthenic type antimonials are valuable. In the case of an adult, a third to half of 1 grain of tartar emetic or 1 to 2 drachms of antimonial wine may be given every three or four hours until relief is afforded. Antimonials should not be given where there is gastric disturbance, prostration, or a feeble pulse. Stimulating expectorants should replace the depressants when sweating occurs and there is free expectoration. When there is extreme dyspnœa and lividity, indicating a distended right heart, venesection or leeching may give great relief. In the same class of case oxygen inhalations greatly relieve the breathing, but the gas should be warmed by passing it through warm water before it is breathed by the patient. In very severe cases the oxygen may be administered continuously, in others at intervals, according to the degree of cyanosis and dyspnœa.

In elderly subjects with signs of prostration and failing heart, diffusible stimulants should be given, ammonia, ether, dry champagne, and old brandy being the most suitable.

During convalescence good food and abundant fresh air are of great importance. Cinchona, nux vomica, and iron, especially in the form of mist. ferri. co., are useful tonics. The best remedy at

this stage, however, is change of air ; and among the most suitable places for the patient to go to are those which are subsequently mentioned as beneficial for cases of chronic bronchitis.

CAPILLARY BRONCHITIS

Symptoms.—Capillary bronchitis, also called “suffocative catarrh,” and by older writers “peri-pneumonia notha,” is the most serious form of bronchitis, involving as it does the bronchial tubes of the smallest size. In young children it is usually complicated with broncho-pneumonia. It may arise by extension, the inflammation travelling downwards from the larger tubes, or it may commence more or less suddenly in the smaller tubes themselves. When it begins suddenly, the earliest symptoms may be rigors in adults or convulsions and delirium in young children. The later symptoms of the illness differ from those of ordinary bronchitis by their greater intensity. There is a comparatively high degree of pyrexia in the case of young children. There is always severe dyspnœa and often orthopnœa. The *alæ nasi* may be seen to actively dilate, and the respirations are always much accelerated and are often irregular. The upper part of the chest is distended, while the lower ribs and the lower part of the sternum are markedly retracted in young children. On auscultation sibilant rhonchi and fine râles are audible all over the chest. As the patient gets worse, the breathing becomes more and more shallow. Cough is frequent and extremely harassing, but expectoration may be absent or very scanty. Both dyspnœa and cough may have paroxysmal exacerbations. The pulse is very rapid, and may be from 140 to 200 per minute. Cyanosis is usually a marked feature, and in children is accompanied with pallor. There is sweating of the upper part of the body, and there is often much restlessness and distress. In adults delirium is common, and in children coma, with twitchings and carpopedal convulsions, is apt to supervene early. In old people there is much prostration. The face at first may be flushed, but this is soon succeeded by pallor. The cough is short and hacking, and the breathing is very rapid. There are often exhausting perspirations, the appetite is in abeyance, the tongue is dry and brown, and there is usually flatulent distension of the abdomen. The patient as a rule soon becomes semi-comatose and delirious.

The **prognosis** is always very grave, and is especially so in the old, in young and rickety subjects, and in the presence of marked cyanosis, delirium, and coma ; but even when the symptoms are

extremely severe, improvement may set in and the patient may recover. Relapses are apt to occur. A severe attack may be fatal in an infant in twenty-four hours, but in adults the illness seldom ends in death before the fourth day.

Treatment.—The treatment of capillary bronchitis differs in few particulars from that of the ordinary form. It must be particularly borne in mind that opium or morphine is usually dangerous. Emetics are very useful at the commencement of acute symptoms. Antimony (gr. $\frac{1}{2}$), ipecacuanha wine (ʒij), or apomorphine (gr. $\frac{1}{2}$) may be given separately or in combination to an adult. For children ipecacuanha wine is the best emetic. Leeching should be adopted when there is extreme dyspnœa with cyanosis, and inhalations of oxygen are also indicated under the same circumstances. Stimulants should be given when there is much prostration.

CHRONIC BRONCHITIS

Symptoms.—The symptoms of chronic bronchitis are persistent cough, with more or less expectoration, wheezing breathing, and dyspnœa, but the malady varies much in degree and in character in different cases. In practice several distinct clinical types are met with.

(1) “Chronic winter cough.”—In the milder forms of this very common variety of chronic bronchitis, the patient, on the advent of cold weather, is troubled with a slight cough, and brings up without much difficulty a moderate amount of mucous or muco-purulent sputum. The cough remains perhaps during the whole of the winter, disappears in the spring, and returns again with the cold weather. Dyspnœa, unless from associated emphysema, is not a marked symptom, and there is little or no soreness of the chest. In the course of time the symptoms increase in severity.

(2) After repeated attacks of acute bronchitis, or after suffering for some years from chronic winter cough, a more severe form of chronic bronchitis supervenes, which is usually associated with emphysema. In this variety the cough is more violent and more persistent, and is especially troublesome in the morning. The expectoration is sometimes scanty, viscid, and difficult to bring up; sometimes abundant, muco-purulent, and easy. In the latter case the sputa may be yellowish-green and typically nummulated. There is often pain or soreness after the cough. Dyspnœa is a marked symptom, especially on exertion. In exacerbations the sputa may be tenacious

and frothy, or diffuent and puriform. In some cases the appetite is bad, the sleep is apt to be broken, and there is bodily wasting, but the temperature remains normal.

(3) The term "dry catarrh" has been applied to a form in which there is troublesome often paroxysmal cough, tightness of the chest, and oppression of breathing, and the expectoration, which is scanty and consists of small, gray, pearl-like pellets, is brought up with difficulty. Dyspnoea is a feature of this form of chronic bronchitis, and usually depends on the coexistence of emphysema. Acute catarrhal bronchitis may occur from time to time.

(4) By "bronchorrhœa," or "chronic pituitous catarrh," is signified a variety of bronchitis characterised by copious expectoration which is watery or glairy, often frothy, and sometimes streaked with blood, or mingled with muco-purulent masses. The cough and expectoration usually occur in paroxysms, during which there is much dyspnoea, while at other times the breathing is easy. This form, as a rule, does not arise independently, but usually supervenes on long-continued bronchitis of the ordinary type. It occasionally persists for a long period without marked deterioration of health. In many cases, however, after a time dyspnoea becomes constant, and progressive wasting and failure of strength occur.

(5) What has been called "gouty bronchitis" is an obstinate form of bronchitis which occurs in gouty subjects, either with or without other manifestations of gout. There are frequent exacerbations, with sometimes severe dyspnoea, cardiac irregularity, and hepatic and gastric disturbance. As the result of intense pulmonary congestion, the sputa may be tinged with blood. The urine is concentrated, loaded with lithates, sometimes deposits uric acid crystals, and may contain albumen.

(6) "Fœtid bronchitis."—Fœtor of the sputa is rarely present in chronic bronchitis unless there is bronchiectasis, but it sometimes occurs independently, usually as the result of an acute exacerbation. Fœtor of the breath not uncommonly accompanies and sometimes precedes the fœtor of the sputa. In character the sputa closely resemble those of bronchiectasis. Fœtor may be a transitory symptom, but it usually indicates a severe form of the disease, and is accompanied by pyrexia, together with loss of appetite and prostration.

Physical signs.—The physical signs of chronic bronchitis are similar to those of the acute form. Emphysema is frequently present, and gives rise to its characteristic signs. When chronic bronchitis starts in early life, pigeon-breast may be produced. In

the slighter forms of winter cough there may be no adventitious sounds, or only a little wheezing, but in the ordinary type the respiration is harsh, the expiration being prolonged and raised in pitch, and sibilant and sonorous rhonchi are usually present, the sonorous rhonchi occurring when the large tubes are affected, the sibilant when those of a smaller size are involved. In cases where the secretion is thin and abundant, crepitant râles may be added to the rhonchi, and are most frequently audible at one or possibly both bases.

Diagnosis.—Some other malady is more commonly mistaken for chronic bronchitis than chronic bronchitis for another disease. Chronic bronchitis is often associated with chronic phthisis, which may be recognised by careful physical examination and by investigation of the sputum for tubercle bacilli. A mediastinal growth or an aneurysm of the arch of the aorta, which presses on the trachea or one of the bronchi, gives rise to symptoms like those of chronic bronchitis, but with dyspnœa of a more severe type and more paroxysmal character than the physical signs would lead one to expect. It is important to distinguish between the primary form of chronic bronchitis and those forms which are secondary to disease of the heart, kidneys, and arteries.

Prognosis.—As a rule, chronic bronchitis tends to get worse with successive winters, but the progress may be slow. The cases which occur in youth or early adult life are the most hopeful. When the malady comes on in old age, it is seldom that it is again got rid of. The most serious forms are those accompanied either with emphysema, which tends to supervene in all cases of long standing, or with degenerated arteries, or with chronic disease of the heart or kidneys. Nocturnal dyspnœa, orthopnœa, loss of weight, and tricuspid regurgitation are all bad signs. The prognosis is better if the patient be in a position to obtain the benefits of change of climate.

Treatment.—In the treatment of chronic bronchitis an endeavour should be made to improve the general health. Fresh air is of great importance, and the patient should be encouraged to lead an open-air life. Overheated and badly ventilated rooms must be avoided. Chill to the surface of the body should be prevented by suitable clothing. The skin should be kept in good condition by baths, and the judicious use of the Turkish bath is often highly beneficial. Bathing the chest with warm water and vigorous friction with a flesh brush, followed by cold douche and rubbing down with a rough towel, may, in certain cases, be employed with

advantage. Stimulating liniments, such as lin. terebinth. or lin. terebinth. acet., well rubbed into the chest at bedtime, are often useful. During exacerbations the treatment appropriate to acute bronchitis must be carried out.

Patients who are plethoric or obese will greatly benefit by a judicious change in the dietary. Inquiries should be made about alcohol, and if there be evidence of excess it should be reduced in amount, altered in kind, or forbidden altogether. The bowels must be carefully regulated. In the case of the secondary forms of chronic bronchitis, treatment should be specially directed to the primary disease.

Cod-liver oil is a remedy which helps to improve nutrition and to bring about a healthier condition of the bronchial mucous membrane. Tonics are useful when the expectoration is profuse, especially quinine, strychnine, perchloride of iron, or mist. ferri co. In the dry catarrhal type quinine may sometimes be given with benefit in a single large dose at bedtime without causing unpleasant physiological effects.

Ipecacuanha and iodide of potassium in combination with carbonate of ammonium may be given when the cough is difficult and the expectoration is scanty, as in the following mixture :—

R Pot. iod. gr. xxiv.
Amm. carb. gr. xxxij.
Vin. ipecac. ʒj.
Syr. prun. Virgin. ʒss.
Aquam ad ʒviij.

Ft. mist. Sig.—An eighth part to be taken three times a day.

Apomorphine in doses of one-tenth of a grain is another useful remedy when the secretion is scanty and the cough troublesome. A convenient formula is :—

R Syrup. apomorphin. hydrochlor. (B.P.C.) ʒij.
Syrup. limonis ad ʒiij.

M. Sig.—A teaspoonful every four hours.

Chloride of ammonium (10 to 20 grains) or carbonate of ammonium (3 to 5 grains) may be given with squill or senega when there is free secretion, as in the following mixture :—

R Ammon. chlorid. gr. clx.
Vin. ipecac. ℥lxxx.
Tr. scill. ʒij.
Ext. glycyrrhiz. liq. ʒj.
Infus. seneg. ad ʒviij.

Ft. mist. Sig.—An eighth part to be taken three times a day.

When cough is distressing by its frequency, relief may be afforded by giving some preparation or derivative of opium, such as tr. camph. co., Dover's powder, morphine, codeine, or heroin. When expectoration is abundant and yellow, balsamic remedies, such as tincture of myrrh (15 minims), balsam of Peru (20 minims), or syrup of tolu (1 fl. drachm), may be added to a mixture; or oil of copaiba (15 minims), myrtol (2 to 5 minims), benzol (5 to 10 minims), oil of turpentine (10 minims), pure terebene (10 minims), or terpene hydrate (2 to 6 grains) may be given in capsules. Tar (syrupus picis liq., U.S.P., 1 to 2 drachms) and garlic (syrupus allii, U.S.P., 2 to 4 drachms) are also useful in the same class of cases. Tar may also be given in the form of perles ($2\frac{1}{2}$ to 5 grains) or as tar-water (1 in 200), dose 5 to 10 ounces, or in the following combination:—

Rx Picis liq. gr. xx.
Pulv. ipecac. co. gr. xxx.
Pulv. benzoin. q.s.

Ft. pil. xxv. Sig.—One pill to be taken every six hours.

Benzo, may be given in the form of guttæ:—

Rx Benzol pur. ʒjss.
Ol. menth. pip. ʒss.
Ol. olivæ ʒij.

M. Sig.—Ten to thirty drops on sugar every three or four hours.

In cases of bronchorrhœa and in old subjects with emphysema, ammoniacum is an efficient remedy. It may be given in the following form:—

Rx Ammon. chlorid. gr. clx.
Liq. ammoniæ ʒij.
Tr. lavand. co. ʒss.
Mist. ammoniaci ad ʒviij.

Ft. mist. Sig.—A tablespoonful to be taken with half a wineglassful of water three or four times a day.

Sedative steam inhalations, such as vapor benzoini, or vapor lupuli, or stimulating, such as vapor terebinthinæ or vapor pini (1 drachm of either tr. benzoin. co., lupulin, ol. terebinth., or ol. pini to 1 pint of water at 140° F.), used for half an hour night and morning, are valuable, the sedative when the cough is tight and the expectoration scanty, the stimulating when the secretion is more abundant. Spray inhalations, such as solution of chloride of ammonium or sodium (1 per cent) are also beneficial in some cases.

Change of air and climate is of much value in the cases where

the bronchitis recurs with the onset of the winter. Great benefit has often followed from a residence during the winter months at Bournemouth, Torquay, Falmouth, or Penzance. Mentone, San Remo or Nervi, Arcachon, Pau, Algiers, Madeira, and Assouan or Luxor are suitable for those who prefer to go further afield.

A course of the mineral waters of Harrogate, Aix-la-Chapelle, Ems, Soden, Mont Dore, Eaux Bonnes, or Cauterets sometimes proves serviceable. Waters which contain sulphur or chloride of sodium appear to be the most suitable. Corpulent patients derive benefit from a course of treatment at Marienbad, Kissingen, or Homburg during the summer months.

PLASTIC BRONCHITIS OR FIBRINOUS BRONCHITIS

The morbid anatomy and pathology of this very rare disease, otherwise called "bronchitis pseudomembranosa" or "bronchial croup," have been described at pp. 63 and 64.

Symptoms.—The malady may show itself in an acute or in a chronic form, but the former is exceptional. The essential feature in either case is the occurrence of paroxysms of cough and dyspnoea, towards the termination of which mucus containing translucent-looking pellets or roundish masses is expectorated. These pellets, when floated in water, are seen to consist of mucus in which are embedded casts of the smaller bronchial tubes rolled up in a sort of ball. The casts are usually tubular, except in the case of those derived from the smallest bronchioles. They vary in size from tiny fragments to masses of 6 or 8 inches in length when unrolled, are usually whitish or pearly gray, and are coated or filled with mucus and in some cases blood. The larger masses sometimes show multiple ramifications terminating in delicate rootlike processes with slightly bulbous ends, forming exact moulds of the bronchial tree to its finest divisions. The casts not infrequently exhibit nodular swellings, which are usually attributed to bubbles of contained air. It is generally stated that the casts are fibrinous, but recent investigations go to show that in some cases at any rate they are chiefly composed of mucin. Some observers have concluded that they are composed of syntonin or coagulated albumen. When seen in section under the microscope they have a fibrillated or stratified appearance. The ground substance may contain leucocytes for the most part of the mononuclear type and alveolar epithelial cells in its interior, and sometimes there are

blood-corpuscles in the outer layers. Separate whorls and cylinders have been observed within an outer laminated layer, producing the impression that an exudation starting in the smaller bronchi has been gradually pushed up into a larger bronchus, where additional exudation has been deposited. Curschmann's spirals and Charcot and Leyden's crystals have been found to be present in some cases where the casts have been small, possessing very few if any branchings. Streptococci, staphylococci, and pneumococci have sometimes been found in the casts; but as these micro-organisms are also present in the purulent secretion of bronchitis, they can have no direct causal relation. The relative number and actual quantity of casts vary greatly. The mucous expectoration may be scanty or abundant, and contain only a few fragments or casts in great numbers. Hæmoptysis is not common, but may occur either before or after the expectoration of the casts, the latter being the more usual. Great relief follows the bringing up of the casts, and during the intervals between the paroxysms the patient is comparatively comfortable and but little troubled with either cough or dyspnœa. The length of the interval varies from a few hours to two or three days, except in the case of the relapses, where it is greater.

The **physical signs** in many cases do not differ essentially from those of ordinary bronchitis. Rhonchi and coarse râles may be generally audible. Deficient movement and suppression of breath sounds, with resonance on percussion, or in some cases dulness due to collapse, will probably be found over a part of the lung, the bronchi of which are plugged with casts. With the expectoration of casts the breath sounds and movements return. Sometimes before the expectoration of casts a peculiar coarse clicking sound has been observed at one or both bases. The sound has been called "bruit de drapeau," or "ventilgeräusch," and is probably produced by the flapping to and fro of loosened portions of cast during respiration.

In acute cases fever is one of the earliest symptoms, and its range is high. There is great dyspnœa, with a feeling of pain and soreness in the front of the chest; the cough is, at first, dry and loud, and the casts are few in number, the illness possibly proving fatal before the characteristic sputa have appeared. In favourable cases, after a few days or a few weeks the pyrexia subsides, and with the expectoration of casts the accompanying dyspnœa ceases and the patient recovers. Sometimes the patient never has another attack, but in other cases the acute form passes into the chronic.

In chronic cases, the expectoration of casts is usually preceded for a longer or shorter period by symptoms of bronchial catarrh, which is apt to persist between the attacks. The attacks themselves are similar in character to those of the acute form, but dyspnoea is usually less intense, and a feeling of oppression and tightness is more common than actual pain in the chest. There is little if any elevation of temperature or constitutional disturbance, while nutrition may be for a time well maintained. The chronic cases are usually of long duration, and extend over a period of years. The relapses in such cases may appear at intervals varying from a week or a fortnight to many months or even years. Between the attacks when large casts are brought up, small or incomplete casts may be spat up with little difficulty, and small fragments may appear for long periods before complete casts are seen. The condition gradually tends to get worse, and the patient loses flesh and strength, but in exceptional cases attacks have recurred for years without obvious deterioration of health. In some cases the illness is preceded by pleurisy, or is associated with pulmonary tuberculosis, and emphysema is a not uncommon sequel. Dropsy, epistaxis, diarrhoea, and albuminuria have been observed as complications.

Diagnosis essentially depends on the discovery of the characteristic casts in the expectoration. In many cases the presence of these may be obvious, but in other cases, unless specially searched for, they may be overlooked. The occurrence of a paroxysm of cough and severe dyspnoea relieved by expectoration is suggestive, and the sputa should be examined by suspension in water. Moulded blood-coagula may resemble the casts of plastic bronchitis, but the facts that they are homogeneous and not stratified, and that they contain blood-corpuscles which are not limited to the outer layer, should serve to distinguish them.

The **prognosis** is unfavourable in acute cases when the symptoms are severe, fifty per cent of the cases proving fatal. In chronic cases, while death rarely occurs as the direct result of the disease, a permanent cure is unlikely.

The **treatment** during the attacks must be carried out on the general lines recommended for bronchitis. Emetics are indicated where there is great dyspnoea and delayed expectoration. The special remedy which has proved most successful is iodide of potassium in 5 to 20 grain doses. Valerianate of guaiacol in 5 to 10 minim doses internally is useful in chronic cases. Inhalations of lime-water, either B.P. strength or diluted with an equal part of

water, and inhalations of 2 to 5 per cent solutions of the carbonate or bicarbonate of soda from an atomiser, and intratracheal injections of glycerin (3i ter die), have also proved beneficial. Mercurial inunctions and, in the chronic form, the coal-tar creosote vapour used in cases of bronchiectasis have been recommended.

BRONCHIECTASIS

The anatomical changes and the causes of bronchiectasis or dilatation of the bronchi have been considered at pp. 77-80. While, as there mentioned, it is seldom a primary disease, it often leads to characteristic symptoms and clinically constitutes a distinct malady.

Symptoms.—The principal symptom is cough, occurring at comparatively long intervals and accompanied by profuse and often foetid expectoration. The cough is usually paroxysmal, and is frequently induced by changes of posture, such as stooping or turning from one side to the other, especially on waking in the morning. The expectoration is usually profuse, a large quantity being brought up at one time, and during the act of coughing it may pour out of the mouth. The probable reason why cough with profuse expectoration thus occurs at intervals is that the secretion lies in tubes which have lost their sensibility, and it is not until there is a sufficient accumulation to reach still sensitive bronchi that expectoration takes place. The sputum in a typical case is greenish in colour, thin and diffuent, and on standing separates into several layers, frothy at the top, then a watery fluid in which muco-pus is suspended, and pus and granular detritus at the bottom. In the deposit may be seen under the microscope, besides granular debris, leucocytes, degenerated epithelial cells, tufts of fungi, and numerous organisms. Sometimes peculiar soft grayish-yellow bodies, from an eighth to half an inch long, may be picked out. These are known as "Dittrich's" or "Traube's plugs," and consist of bundles of leptothrix filaments, with leucocytes, red blood-corpuscles, crystals of hæmatoidin and fatty acids, and fatty epithelial cells. Fœtor both of the sputa and of the breath is usually present, sometimes to an intense degree. These symptoms are most marked in basic cases.

Hæmoptysis is not uncommon at some stage of the disease, but a large hæmorrhage does not usually occur until the dilatations are of considerable size. A slight degree of dyspnœa is often experi-

enced, but is not as a rule very noticeable except on exertion. In the later stages, and when bronchitis and emphysema complicate the bronchiectasis, dyspnœa may be a very distressing symptom.

The general nutrition is often good, and frequently the patient has, to the casual observer, the appearance of health. In most cases, at least a slight degree of cyanosis may usually be observed in the lips, hands, and cheeks, and clubbing of the fingers and toes is an almost constant feature and may attain an extreme degree.

In the later stages the usual signs of chronic congestion in the liver, spleen, and kidneys show themselves, and dropsy is likely to supervene. Albuminuria from lardaceous disease of the kidneys, or chronic diarrhœa from the same affection of the intestines, or from septic infection, may occur. Pyrexia of a remittent type is not uncommonly a late symptom, and with that the nutrition and strength fail.

Physical signs are sometimes indefinite, resembling those of bronchitis, and, when characteristic, vary according to the extent of the disease and the degree of fulness or emptiness of the tubes. When the dilated tubes are full, there is dulness with feeble breath sounds, or distant tubular breathing over the affected area. When the tubes are partially or completely empty, the dulness may be replaced by high-pitched resonance; the breath sounds are tubular or cavernous; and coarse, often metallic, râles are audible on auscultation.

There are usually signs of contraction of the affected part of the lung, there being diminution both in the size and movements of the corresponding part of the thorax. The heart is displaced and is drawn over to the affected side, the impulse being sometimes felt to the right of the right nipple line when the seat of the disease is the right base, or in the left anterior axillary line when the left base is affected.

Prognosis.—The disease is usually one of slow progress and long duration, and the patient probably succumbs in the end to some complication or intercurrent affection, such as hæmoptysis, bronchitis, broncho-pneumonia, pneumonia, pulmonary gangrene, or cerebral abscess, the latter being a mode of termination which is not very rare. When once the condition of bronchiectasis has become well marked, recovery is out of the question, but much can be done by judicious treatment to arrest or slow further progress.

Diagnosis.—The combination of profuse, probably fœtid, expectoration, coughed up at comparatively long intervals with

physical signs such as have been described, is sufficiently characteristic. A striking confirmation of the diagnosis may sometimes be obtained by partial inversion of the patient, which in basic cases is usually followed by cough and the emptying of the secretion in the dilated bronchi.

In gangrene of the lung there is foetor, both of the breath and sputum, but not as a rule the paroxysmal cough with profuse expectoration, while there is pyrexia and a greater degree of prostration. Examination of the sputa usually shows the presence of pulmonary tissue, which is not found in cases of bronchiectasis.

From foetid bronchitis the diagnosis depends on the presence or absence of physical signs of dilated tubes. From empyema opening into the lungs the diagnosis must be made by means of the history and physical signs (p. 259). From phthisis the diagnosis is only difficult when the disease is situated in the upper part of the lungs, in which case the absence of tubercle bacilli and of elastic fibres and the apyrexial course are important points.

Treatment.—Much of what has been said with regard to the treatment of chronic bronchitis applies with equal force to the treatment of bronchiectasis. Special attention should, however, be directed to securing the regular evacuation of the secretion. One of the most effective methods of doing so is to get the patient to partially invert the body by hanging over the edge of his couch or bed and resting his hands on the floor, by which means, as already mentioned in connection with the diagnosis, cough is generally produced, and the secretion, if at all profuse, comes freely away. This should be practised two or three times a day, on waking in the morning, about mid-day, and before going to bed at night. This plan of treatment alone will sometimes bring about very great improvement in the condition of the patient and a striking diminution in the amount and in the foetor of the expectoration. The most useful drugs when foetor is a marked symptom are garlic, tar, and asafoetida. Garlic may be given in the form of *syrupus allii* (U.S.P.) (dose 2 to 4 drachms for an adult), or as a cachet containing 20 to 60 grains of the powdered drug. *Syrupus picis liquidæ* (U.S.P.) (dose 1 to 4 drachms) and *spir. ammon. foetid.* (dose $\frac{1}{2}$ to 1 drachm) may be given, either alone or in combination with garlic.

Intratracheal injections administered by means of a syringe with special nozzle which can be passed into the larynx between the cords have been recommended. An oily solution consisting of ten parts of menthol and two parts of guaiacol dissolved in eighty-eight parts

of olive-oil was first employed, one drachm being injected into the trachea twice a day. Better results have, however, recently been obtained by employing glycerin as the vehicle, and izal (in a proportion of 10 per cent) as the agent.

One of the most beneficial methods of treatment, especially in the case of adults, is that by means of the vapour of coal-tar creosote. The treatment is, however, somewhat difficult to carry out, except at a hospital, as a room must be set apart for the inhalations. The vapour, which is produced by heating a few ounces of the liquid coal-tar creosote in a metal saucer over a spirit-lamp, is very irritating to the mucous membranes, and acts by producing cough and expulsion of the retained secretions. Under this treatment, in favourable cases, the amount of the expectoration greatly diminishes, the foetor sometimes entirely disappears, and the patient improves very considerably in health.

STENOSIS OF THE TRACHEA AND BRONCHI

The causes of stenosis of the trachea and bronchi have been considered at p. 85.

Symptoms.—The main symptom of stenosis of the trachea is dyspnoea, which, while usually present, varies in degree from time to time as the result either of temporary swelling of the mucous membrane or of pressure on the laryngeal nerves. The breathing is difficult and laboured, the auxiliary muscles of respiration are called into action, and there may be inspiratory retraction of the lower part of the thorax. The difficulty of breathing is increased when the patient lies down. The breathing is slow, both inspiration and expiration being prolonged. The inspiration is more laboured than the expiration and is accompanied by stridor, and there is sometimes a peculiar “mewing” inspiratory murmur, which is most marked on exertion. The voice may be weak, but is otherwise unaffected. Cough is often present from the tracheal irritation or from accumulation of mucus below the swelling. In some cases it may be observed that the pulse becomes weaker or may disappear altogether during inspiration (*pulsus paradoxus*). It was pointed out by Gerhardt that in laryngeal stenosis the larynx makes wide excursions during respiration, and the head is usually thrown backwards, while in tracheal stenosis the movements of the larynx are only slight, and the head is bent forwards or held in the ordinary position. In bronchial stenosis dyspnoea is present, but to a lesser

extent than in tracheal. The respiratory movements and the circumference of the affected side are diminished, while the opposite side is over-distended. There are feeble breath sounds, accompanied by sibilant and sonorous rhonchi over the affected part, while the percussion note remains clear.

Treatment.—In cases of severe dyspnœa from stenosis of the trachea, tracheotomy should be performed, provided it be possible to make sure that the trachea can be opened below the obstruction. In cases of syphilitic stenosis this can seldom be done. Steam inhalations of hop and benzoin, inhalations of oxygen, or subcutaneous injection of strychnine and ether may give temporary relief both in tracheal and bronchial stenosis. In cases where there is marked venous congestion, venesection should be performed. Morphine injections or the inhalation of chloroform may be tried in cases of great distress.

FOREIGN BODIES IN THE AIR-PASSAGES

The pathological effects of foreign bodies in the air-passages have been considered at p. 54.

Symptoms.—When a foreign body lodges in the larynx or trachea the patient is seized with a paroxysm of dyspnœa and a feeling of suffocation, accompanied with violent coughing. Death may shortly ensue from suffocation, or the paroxysm may subside after a time, but probably only to return, until relief is afforded by the surgeon. If the body be in the larynx, the voice will be altered or may be lost altogether. When a foreign body lodges in a bronchus, the initial symptoms vary according as the body is rounded, smooth, and unirritating, or rough, angular, and spiky. If smooth, no immediate effects may be observed; if rough, spasmodic cough or paroxysmal dyspnœa resembling asthma may occur. Hæmorrhage is likely to follow the introduction of a sharp pointed body. The violent coughing may induce vomiting. Expectoration at first is frothy and mucous, but later becomes muco-purulent. A sense of soreness or actual pain may be felt behind the trachea.

When the foreign body is in the larynx or trachea, the physical signs are those of impeded entrance of air into the lungs. When it is lodged in a main bronchus, the respiratory movements of the corresponding lung are diminished, the percussion note is unaltered, the breath sounds are diminished and probably accompanied by rhonchi, and the vocal fremitus is weakened.

If secondary pneumonia, bronchiectasis, or abscesses develop, these will give their characteristic symptoms and signs.

Examination by means of Röntgen rays may show exactly the position of the foreign body.

The **diagnosis** depends on the history, combined with the symptoms and physical signs. There is usually a history of a foreign body having been in the mouth, and having been inhaled or gone down the wrong way. Sometimes the body has been thought to have been swallowed, and symptoms have not been observed for a considerable interval.

The **prognosis**, as a rule, is unfavourable, unless the foreign body be removed or come away; but cases have been recorded where a foreign body, such as a half-sovereign, has remained for many years lodged in a bronchus without giving rise to any symptoms.

Treatment.—An attempt should be made to remove the foreign body, where practicable, as soon as possible. Tracheotomy should be performed when the foreign body is in the larynx and producing suffocative dyspnœa, or when it is in the trachea or one of the main bronchi. The foreign body may then be extracted by forceps if within reach, or the patient may be inverted and shaken in order to dislodge the body when in the bronchus. In chronic cases, with formation of cavities or abscesses, these should be opened, and exploration made as far as possible with probes and forceps, by which means the foreign body may sometimes be removed.

ASTHMA

Asthma is a disease characterised by paroxysmal attacks of dyspnœa, during which the respiratory movements are much diminished. It is sometimes spoken of as “spasmodic asthma” or “bronchial asthma,” but the latter term is limited by some authorities to the form associated with emphysema and chronic bronchitis. The term asthma is popularly used as synonymous with dyspnœa, as when we speak of “cardiac asthma” or “renal asthma.” Hay fever is sometimes called “hay asthma” (see p. 40), but true asthma may be excited by hay, etc.

A first attack of asthma most usually occurs under the age of ten or between twenty and forty-five. The predisposing causes have been considered in the section on general etiology, pp. 50-56. The immediate exciting causes may be classified as follows:—

(a) Central. Effects of emotional disturbance, such as are produced by fright, grief, anger, etc.; of imagination, as when an attack is induced by sleeping in a dark room; or of exhaustion from over-exertion.

(b) Pressure on the vagus by enlarged bronchial glands, tumours, etc.

(c) Reflex, through impressions on distant organs, or other parts of the body, including cold to the surface, flatulence, distension of the stomach, or the presence in it of food either in itself indigestible or with regard to which some idiosyncrasy exists, loading of the rectum, uterine disorders, menstruation, pregnancy, and parturition, nasal disorders.

(d) Irritation of the mucous membrane of the upper respiratory tract, or of the bronchi by certain scents, such as flowers, hay, and ipecacuanha, and by the emanations of certain animals, rabbits, cats, hares, etc., or by various kinds of dust, foreign bodies, volatilised lead oxide, sulphurous acid, nitrous oxide, chlorine, fogs, and air possessing peculiar qualities depending on special conditions of the atmosphere. Forcible respiratory movements, such as laughing, sneezing, or coughing, may induce an attack, and possibly act by stimulating the bronchi.

(e) Blood states, such as gout.

Pathology.—The following are the main theories which have been put forward to explain the direct cause of the asthmatic paroxysm:—

(1) The spasm theory, that the attacks essentially depend on spasm of the circular muscular fibres in the smallest bronchi.

(2) That the attacks depend on swelling of the mucous membrane of the smaller bronchi, either of an inflammatory nature, due to microbic infection or acute catarrh, or of the nature of urticaria and due to dilatation of vessels from vasomotor influences.

(3) That they depend on centripetal irritation of the vagus, causing spasm of the diaphragm, intercostals, and other muscles of inspiration.

(4) That they depend on spasm of the diaphragm only.

(5) That they are due to paralysis of the bronchial muscles.

The spasm theory of the causation of asthma is that which is most generally accepted. We have no absolute proof that spasm of the bronchioles will cause dyspnoea, but the theory is supported by the fact that relief to the paroxysm is afforded by such remedies as stramonium and belladonna, which paralyse unstriated muscular fibre, and is further borne out by experiments of Paul Bert, which

showed that stimulation of the pneumogastrics will cause contraction of the muscular coats of the small bronchi. The spasm theory, however, will not account for the catarrh which is associated with the dyspnœa.

The peculiar characters of the expectoration, the presence of spirals, crystals, etc., point to a catarrhal affection of the small bronchi; and Curschmann, influenced by this, suggested the term "bronchiolitis" as specially appropriate for the affection.

The phenomena of spasm of the diaphragm differ *in toto* from those of asthma, and fixed inspiratory distension of the thorax is far from being a constant feature. The theories referring asthma to these causes may therefore be dismissed, as may also that attributing it to paralysis of the bronchial muscles. While we cannot be certain as to the precise cause of the paroxysm, it is probable that both spasm of the bronchiole muscle and catarrhal swelling of the mucous membrane occur; but that disordered innervation is an important factor in the etiology is shown by the part played by indirect agencies acting reflexly, and by the association of asthma with nervous disorders in general.

Symptoms.—An attack of asthma is not infrequently preceded by certain premonitory symptoms or so-called "aura asthmatica." These vary in character in different cases, and may continue for a few hours only or for several days. Before the onset the patient may be unusually buoyant, or depressed or irritable; he may feel drowsy or disinclined for exertion; he may suffer from headache, neuralgia, or giddiness; sometimes he complains of chilliness or itching under the chin and between the shoulders, or he has a severe fit of sneezing; his face may flush all over or on one side only; or he may be troubled with dyspepsia or have profuse diuresis. In some cases the attack itself commences with a feeling of constriction in the thorax, a dry cough, and a moderate degree of dyspnœa.

Occasionally the paroxysm begins during the day, but, more commonly, it comes on during sleep, the patient waking up in the early hours of the morning with a feeling of suffocation which causes him to fight for his breath. In a severe attack, the expression is one of extreme anxiety, the face flushed or pallid or dusky, the eyeballs protruding, the conjunctivæ watery and suffused, the nostrils dilated, and the mouth open and gaping. While the extremities are cold, perspiration breaks out over the trunk and the face and neck. So fully occupied is the patient with his respiratory difficulties that he has no breath to spare for speech, and if spoken to probably answers by signs only, or in monosyllables. So great is the desire

for air that sometimes the patient will bare his chest and sit by an open window even in the coldest weather. The attitude he assumes is characteristic. The head is thrown back or together with the body is bent forward, while the shoulders are raised and the arms are supported on the knees or on a desk or table. The chest is fixed in the position of full inspiration; the diaphragm is depressed; the abdominal muscles are tense and the auxiliary muscles of respiration are brought fully into action.

The respirations are noisy, slow, and shallow, expiration especially being unduly prolonged in the ineffectual effort to expel the air over-distending the lungs. A normal or subnormal temperature is the rule, while the pulse is small and weak and may be slow and irregular. The urine, at first probably pale and abundant, later becomes high-coloured and scanty, when the attack passes off.

Expectoration does not usually occur until towards the end of the paroxysm. The sputa generally consist of small translucent whitish or glairy pellets like sago or tapioca, sometimes accompanied by similar masses of half an inch or an inch in length. If, however, asthma is accompanied by bronchitis, the sputa are more abundant and muco-purulent. Occasionally in severe attacks the sputa contain blood in more or less abundance, but in our experience this has only occurred in cases where the paroxysm has been due to the pressure of an aneurysm on the trachea or one of the bronchi. Certain constituents of the sputum require special mention. Leyden and Curschmann first drew attention to the presence of certain peculiar spiral bodies, twisted casts of the smaller tubes. These bodies, however, are rather of pathological interest than of clinical importance, and in many cases of asthma they may be searched for in vain. To find them the sputa should be spread out on a dark plate and the round whitish masses picked out, unrolled, and examined under the microscope. The spiral, which is composed of a substance akin to mucin, has a characteristic appearance, and consists of a fine central core with zigzag course, round which is coiled a spirally arranged network of fine threads. Sometimes epithelial cells and crystals may be seen clinging to the surface of the spirals. Curschmann's spirals are not peculiar to asthma, but are occasionally observed in other pulmonary diseases, such as pneumonia and capillary and plastic bronchitis. Other interesting points with regard to the sputum are its richness in eosinophile cells, which are also in excess in the blood, and the occasional presence of peculiar colourless, sharply pointed octahedral crystals, probably

identical with the crystals found in post-mortem blood and in semen. These crystals, which were first described by Leyden and Charcot, are composed of the phosphate of some organic base, probably spermin.

On **physical examination** the signs are, as a rule, those of emphysema. The percussion note is over-resonant and drum-like, and the cardiac and liver dulness may be encroached on or obliterated. The respiratory murmur is temporarily obscured by sibilant or sonorous rhonchi, present, as a rule, on both sides, although in exceptional cases on one side only, or even in parts of the lung.

In the expired air oxygen may be almost entirely absent, being replaced by carbonic acid.

In *duration* the paroxysm varies considerably in different subjects and in different attacks in the same individual, lasting in some cases only thirty minutes or an hour, and in others continuing with slight intermissions for twenty-four or thirty-six hours, or in cases complicated with emphysema and bronchitis persisting with more or less intensity for weeks.

The shorter paroxysms usually terminate suddenly, leaving behind them nothing more than a feeling of drowsiness or of languor and weariness, while the more protracted subside gradually, shortness of breath probably remaining for some time after the actual paroxysm is over.

Attacks may recur at more or less regular intervals. There may be a number occurring in a short time followed by a long period of immunity, or they may come on quite irregularly, and no rule can be stated to serve as a guide in individual cases. In favourable cases the attacks, after appearing at longer and longer intervals, may entirely cease; in other cases, while becoming less severe, they increase in frequency and the sufferer becomes permanently short of breath, his malady being then really chronic bronchitis and emphysema.

In the case of asthma *in childhood* the dyspnoea is usually less definitely paroxysmal and less severe, but is more enduring than in the adult. Children who suffer from asthma are apt to be stunted in their growth and to acquire a stoop, with rounded shoulders and pigeon-breast. In a large proportion of cases occurring in later life a history of asthma in childhood may be traced. A good many of the juvenile cases, however, improve with advancing years, the affection ceasing altogether at puberty.

As a rule asthma in young children is a phenomenon of

bronchitis ; in adolescents, it is more definitely a neurosis ; whilst in older persons it complicates established pulmonary disease, more especially chronic bronchitis and emphysema.

Prognosis.—The younger the patient the greater is the chance that the disease may disappear. After middle life it tends to be progressive, and its association with bronchitis and emphysema is very unfavourable. The paroxysm itself very rarely proves fatal.

Diagnosis.—The diagnosis of asthma is not as a rule a matter of great difficulty. The principal conditions which may be mistaken for asthma are spasmodic dyspnœa due to laryngeal disease, the spasmodic type of acute bronchitis, cardiac dyspnœa, emphysema, and thoracic aneurysm. The dyspnœa resulting from laryngeal disease is mainly inspiratory, and there is retraction of the lower ribs due to the want of expansion of the lungs, contrasting with the over-distension which accompanies the asthmatic paroxysm. In the spasmodic type of acute bronchitis, cough is present from the first, while in asthma a cough only comes on when the paroxysm is declining. In cardiac dyspnœa, the breathing is usually hurried, and signs of cardiac disease are generally to be made out. In thoracic aneurysm, paroxysms of dyspnœa may occur, closely resembling those of asthma. They are common in the case of aneurysms of the transverse part of the arch pressing on the trachea or on one of the main bronchi. Such aneurysms have to be recognised by symptoms rather than by physical signs. The paroxysms are very severe and protracted and do not readily yield to the ordinary remedies for asthma. The age, sex, habits, and occupation, the occurrence of or freedom from similar attacks previously, the presence or absence of tracheal tugging or paralysis of the recurrent laryngeal, are points which may help in diagnosis.

Mediastinal tumours, which may also give rise to paroxysms of dyspnœa, must be recognised by the other symptoms or physical signs which they produce. Röntgen-ray examination may throw light on the nature of a doubtful case.

Treatment has to be directed first to the relief of the paroxysms when they occur, and second to their prevention or to the diminution of their number and severity.

When the paroxysm appears to be connected with a heavy meal or some particular article of food, an emetic, such as 20 grains of ipecacuanha, is specially valuable at the outset. Local applications, such as turpentine stupes or linseed and mustard poultices, may be employed in cases where there is a feeling of constriction or soreness of the chest. Nothing, as a rule, gives so much relief

to the dyspnœa as the inhalation of the fumes from the slow combustion of nitre or powdered stramonium seeds or leaves. A powder largely used for this purpose at the Brompton Hospital consists of two parts of powdered stramonium leaves, one part of powdered nitre, and one part of powdered aniseed. Proprietary remedies, such as Himrod's powder, contain similar ingredients. Medicated cigarettes, such as Joy's or Slade's, some of which contain arsenic instead of or in addition to stramonium and nitre, are also useful.

Morphine internally or hypodermically is a remedy potent to relieve, but it must be used with discretion. Inhalations of chloroform, amyl nitrite, or iodide of ethyl may also be efficacious. Strong black coffee is useful early in an attack. Lobelia, a depressant and nauseant promoting secretion and expectoration, benefits in some cases, the ethereal tincture being given in doses of 5 to 15 minims every half-hour until a nauseating effect is produced. Grindelia robusta, the action of which is somewhat similar to that of lobelia, is similarly employed, 10 to 30 minims of the liquid extract (B.P.C. and U.S.P.) being repeated every half-hour until relief of dyspnœa. Chloral hydrate, belladonna, stramonium, nitro-glycerine, and iodide of potassium have all been found useful when given internally. A convenient formula combining stramonium and iodide of potassium is the following, which is much used at the Brompton Hospital:—

℞ Extr. stramonii, gr. ij.
 Extr. glycyrrhiz. gr. viij.
 Pot. iod. gr. xxiv.
 Sp. chlor. ℥xl.
 Aq. menth. pip. ad ℥viij.

Ft. mist. Sig.—An eighth part to be taken three times a day.

Amongst the vast variety of remedies which have been suggested in the treatment of asthma, the value of any one in any particular case is most uncertain.

In our efforts to prevent the paroxysms altogether, or, failing that, to diminish their number and severity, we must carefully consider each individual case. We must satisfy ourselves that there is no impediment to free breathing through the nose. Obstruction due to polypi, swelling of the turbinate bones, adenoid growths, enlarged tonsils, or other cause must be appropriately treated. The home conditions must be investigated. The bedroom should be airy and well ventilated. If the attacks continue, a change of bedroom, house, or locality should be tried. The diet should be plain

and digestible, all rich or irritating foods being eschewed, and late suppers avoided. Regularity of the bowels must be maintained. Alcohol, except in great moderation, is injurious. Exercise in the open air, especially riding or cycling, is highly beneficial. Baths, in particular shower-baths, followed by friction with a rough towel, and, in the case of confirmed asthmatics, compressed air-baths two or three times a week, are most useful. Oxygen inhalations have been tried, but have not been found beneficial. Change of air and surroundings is often helpful. Torquay, Bournemouth, and Falmouth in this country, Cimiez, Pau, Arcachon, and Mont Dore on the Continent, or Florida and Southern California in America, may be mentioned as suitable resorts for the middle-aged, while St. Moritz or Davos Platz may benefit young subjects with little emphysema.

As a rule, the air of towns suits asthmatical subjects better than the country, but often the individual has to find out for himself the locality which suits him best. It is important, however, to remember that a person may suffer from attacks in one room and be free from them in another in the same house.

Such conditions as anæmia, dyspepsia, or constipation, and any existing bronchitis, should be appropriately treated. Iodide of potassium or ammonium, arsenic, belladonna, atropine, or stramonium may be given internally, singly or combined, in cases where any degree of dyspnoea continues between the paroxysms.

HECTOR MACKENZIE.

DISEASES OF THE LUNGS

PULMONARY EMPHYSEMA

Pulmonary emphysema is a disease which is essentially characterised by a relative increase of the proportion of air space in the lung. It was defined by Laënnec as "an excessive, permanent, and unnatural distension of the air-cells." The anatomical conditions and pathology have been described at p. 80.

Symptoms. — The most constant and most characteristic symptom is shortness of breath, which at first is only noticed on mounting a flight of stairs, on walking up a slope, or on making any rapid movement, or after efforts such as stooping, in which the breath has to be held, or after a heavy meal, when the movements of the diaphragm are impeded. As time goes on, dyspnœa becomes more marked and more and more constant, being aggravated by the slightest exertion, while exacerbations closely resembling asthma are of frequent, sometimes of nightly, occurrence. The sufferer becomes unable to lie down, and has to sleep in a chair or propped up with pillows. A certain degree of cyanosis is usually present, and in some cases is as extreme as in congenital heart disease, and this sometimes before marked dyspnœa has developed. In a typical case the aspect is characteristic: the expression anxious, the cheeks and lips blue, the face bloated, and the eyes prominent and injected.

Cough is a frequent but not a necessary symptom, the emphysematous patient being peculiarly liable to winter cough. The cough is usually attended with the expectoration of small grayish pellets of mucus, and is sometimes of a paroxysmal character. The sputa are sometimes blood-stained, and occasionally large hæmorrhages have occurred. Attacks of bronchial catarrh are induced by comparatively slight causes, and add considerably to the difficulty of breathing. As a rule, it is on account of the bronchitis that treatment is sought, and without this accompaniment one rarely sees emphysema in hospital practice. Dyspepsia and flatulence are common, and are probably due partly to catarrh of the stomach predisposed to by chronic congestion, and partly to insufficient exercise.

The obstruction to the pulmonary circulation resulting from the anatomical changes in the lung is for a time compensated for by hypertrophy of the right ventricle. But after a while this cavity dilates and the muscle is insufficient to completely overcome the obstruction of the obliterated capillaries. Accordingly, in the later stages the symptoms of general venous backward pressure supervene, the dyspnœa and the degree of cyanosis increase, and œdema and general dropsy may be added to the other symptoms. In advanced cases there is marked emaciation and loss of strength.

Physical signs.—When the disease is established the physical signs are characteristic. The thorax is generally increased in size and altered in shape. It is especially enlarged in its antero-posterior and oblique diameters, the increase being proportionately more marked above than below. The attitude is stooping, the shoulders are raised, the back rounded, the sternum prominent and arched, the costal angle widened, and the ribs more nearly horizontal. The term “barrel-shaped” is usually appropriate for the form of the chest, but deformities due to rickets or other states sometimes cause the type described to be departed from. While the size of the chest is increased, its respiratory capacity is much diminished. It will be found on measurement that very little expansion takes place on deep inspiratory efforts. The movements of the chest-wall are principally elevatory, brought about by the action of the accessory muscles.

On percussion the note is drum-like and full-pitched and the area of pulmonary resonance increased, the areas of heart and liver dulness being diminished or altogether obliterated. The lungs overlap the heart, so that the cardiac impulse is not appreciable in the normal situation, while pulsation may be seen and felt in the epigastrium. On auscultation the breath sounds, especially the inspiratory part, are weak, and accompanied by rhonchi if there be bronchial catarrh. The vocal fremitus and resonance are usually diminished. The cardiac sounds are feeble, and a systolic murmur may be heard at the apex of the heart or to the left of the sternum, which in some cases is due to tricuspid incompetence. The pulse is generally small and weak. The diaphragm and liver are depressed.

In advanced cases there may be pulsation in the jugular veins, enlargement of liver, albuminuria, anasarca, and ascites.

Inasmuch as the *atrophic form of emphysema* is a senile change, and only a part of a general wasting of the tissues of the body met with in aged and withered-looking persons, the symptoms are less

characteristic than in the ordinary form. Dyspnœa is little felt, and asthmatic paroxysms are of rare occurrence. There is, however, a considerable liability to bronchitis, and this may be attended with profuse expectoration. In this form of emphysema the thorax, although altered in shape, is rather diminished than increased in size. It becomes rounded, not from an increase in the antero-posterior diameter, but from a contraction in the lateral diameter, the ribs becoming more oblique instead of more horizontal, as in the other form, so that the lower ribs may nearly reach the iliac crest. The sternum is pressed forwards, and the clavicles become markedly convex. The percussion note is drum-like, except at the apices, where the resonance may be impaired without any consolidation being present, and the breath sounds are feeble all over. The heart is not displaced and its area of dulness is not diminished.

Diagnosis.—When the symptoms and physical signs described are present, no great difficulty should be experienced in making a diagnosis. Emphysema is most frequently mistaken for cardiac disease, an error especially likely when a systolic murmur is present. Cyanosis is, as a rule, more marked in emphysema than in any form of heart disease except mitral stenosis and congenital malformation, both of which can generally be readily recognised. The characters of cardiac dyspnœa, moreover, differ from those of emphysema, there being paroxysms of panting breathing brought on by exertion or emotion, during which the respiratory movements are free. It is said emphysema has been mistaken for pneumothorax, or *vice versâ*, but such a confusion could only occur as the result of insufficient examination. An aneurysm of the transverse arch may give rise to symptoms of dyspnœa, as referred to in the diagnosis of asthma. The emphysematous form of pulmonary tuberculosis must be borne in mind and the sputum examined for tubercle bacilli.

Prognosis.—A good prognosis may be given in cases where there are no serious symptoms, provided the patient be placed under favourable conditions, when it is likely that the disease will remain stationary. Should the patient be obliged to continue under the conditions which have produced the disease, it will probably pass into a more serious form. When the disease is advanced the duration of life is unlikely to be long. In the atrophic form of emphysema the outlook depends on the general rather than on the local condition. In such cases the vital power is feeble and life may be cut short at any time.

Treatment.—In early and moderately advanced cases con-

siderable improvement may result if the patient be placed under favourable conditions. Such forms of exercise must be avoided as are likely to put an increased strain on the lungs. The patient must therefore go slowly upstairs or up an incline, and must rest immediately he feels at all out of breath. A course of arsenic and cod-liver oil does good by improving the condition of the blood and the general nutrition. Iron, quinine, strychnine, and the mineral acids are useful tonics, especially in the senile form of the disease. It is very important to cure and to prevent the recurrence of bronchial catarrh, and the measures recommended under the heading of chronic bronchitis may be adopted. Respiratory exercises which promote the compression of the thorax during expiration are useful. A course of compressed-air baths often greatly benefits, diminishing the symptoms and physical signs and increasing the vital capacity. A special chamber, large enough to contain several persons, is used, in which the pressure is gradually increased to $1\frac{2}{3}$ atmospheres. The patient remains in the chamber for two hours, and takes the bath three times a week. Similar but less striking results are obtained by the use of Waldenburg's apparatus, by means of which the patient respire into rarefied air. In advanced cases with urgent dyspnoea and cyanosis, venesection may afford immediate relief, and oxygen inhalations are also very valuable. Asthmatic attacks may be appropriately treated by some of the measures referred to under the heading of asthma. When the heart is failing, digitalis, strophanthus, strychnine, and caffeine are the most useful drugs.

PULMONARY CONGESTION AND ŒDEMA

Pulmonary œdema and congestion have been considered in their anatomical and pathological relations at pp. 73, 74. Pulmonary œdema is usually a secondary affection supervening in the late stages of many diseases as the result of failing heart. In rare cases, however, it comes on suddenly with characteristic symptoms described below. Pulmonary congestion may be either acute or chronic.

Symptoms.—*Acute congestion* is the first stage of acute pneumonia, and, as has been pointed out, it is probable the so-called "acute idiopathic congestion" or "Woillez's disease" is an abortive form of lobar pneumonia. The symptoms which have been

observed in cases of this kind are similar to those of pneumonia. There is an initial chill, which is followed by pain in the side, dyspnœa, cough, and pyrexia. Herpes often breaks out on the face. After two to four days the fever subsides and the symptoms abate. The physical signs are also like those in the early period of pneumonia. Usually at one or other base there is impaired resonance with feeble or sometimes bronchial breath sounds and fine crepitant râles.

In *passive congestion*, which is most commonly secondary to cardiac disease, the symptoms are not characteristic, but dyspnœa and cough are usually present, and the expectoration sometimes contains large pigmented cells, as described at p. 106. In *hypostatic congestion* the symptoms are commonly negative, and its existence is only revealed on physical examination, which shows dulness and feeble or bronchial breathing, with râles at one or both bases. *Hypostatic pneumonia*, which is a later stage of hypostatic congestion, also has no very distinctive symptoms. Sometimes along with it there is a rise of temperature, but sometimes pyrexia is absent. The respiration is usually accelerated. When it occurs in the course of specific fevers there is no cough and no expectoration of rusty sputum. The physical signs do not differ from those of hypostatic congestion. In cases where hypostatic pneumonia supervenes there is usually much weakness, and convalescence is very slow.

The symptoms of the ordinary secondary form of œdema are increasing dyspnœa, cough and watery sputa, with deficient resonance and râles at the bases of the lungs. In the form of acute œdema, which sometimes comes on in cases of pleurisy with effusion after paracentesis, respiratory distress may be little marked, but there is profuse watery expectoration.

The symptoms of *acute idiopathic œdema* are of sudden onset, and consist of dyspnœa, which is often intense, extreme respiratory distress, and incessant harassing cough, with usually very abundant expectoration. The patient is quite unable to lie down, and his respirations are hurried, laboured, and wheezing. The expectoration is watery, frothy, albuminoid, and often limpid, or, if congestion accompany the œdema, of a réddish or salmon colour. Sometimes as much as a pint and a half of secretion is coughed up within a few hours. From apex to base crepitant râles are audible, and the chest is over-distended and more resonant than usual. The temperature is usually normal or subnormal. The pulse, at first full, becomes small, rapid, and weak, the low systemic arterial

tension being in marked contrast with the high pulmonary tension. The face is livid, the eyes are staring, and the veins in the neck turgid. The expectoration, at first abundant, becomes scanty, and in rare cases it may be scanty throughout. The patient usually dies with symptoms of intense asphyxia within one or two days, but sometimes the illness ends in recovery.

Diagnosis.—Acute pulmonary œdema may be mistaken for asthma, cardiac or renal dyspnœa, or pulmonary embolism. The resemblance to asthma is, however, only superficial, for air enters the lungs freely and, although the breathing is quick and panting, there is no expiratory difficulty. Asthma is not attended with watery expectoration. The suddenness and severity of the attack, the character of the expectoration, and the presence of crepitant râles will help to distinguish it from cardiac and renal dyspnœa. The symptoms closely resemble those of pulmonary embolism, but the profuse watery secretion is an important point of difference, while the causes of embolism will probably be wanting.

Treatment.—Acute idiopathic congestion requires the same treatment as pneumonia in its early stage. In passive congestion or secondary œdema, treatment must be directed to the primary disease. Cases of hypostatic congestion require stimulating treatment. In the presence of symptoms of acute pulmonary œdema, venesection should be promptly performed, and the amount of blood withdrawn should be large enough to immediately relieve the respiratory distress and lower the pulmonary tension. On account of the tendency to failure of the heart's action, hypodermic injections of strychnine, gr. $\frac{1}{24}$, with caffeine, gr. ij, should be given. Sal-volatile and spirit of ether or brandy may be given by the mouth, or if swallowing be difficult brandy and beef-tea may be administered per rectum. Sinapisms may be applied over the heart, and the extremities should be kept warm by means of hot-water bottles. Free purgation by means of saline aperients may help to diminish the engorgement of the venous system.

PULMONARY EMBOLISM AND THROMBOSIS

The pathology and anatomical changes produced by pulmonary embolism and thrombosis have been considered at pp. 75-77.

Symptoms.—In some cases the lodgment of an embolus in one of the main branches of the pulmonary artery produces absolutely sudden death. In other cases the immediate effect

is syncope, followed by convulsions which generally shortly terminate in death, but from which in rare cases the patient rallies. Sometimes the first symptom is acute dyspnœa, and this always ensues when life is prolonged. The respirations in this case are quickened, panting, and probably shallow, but the patient is able to breathe deeply. Sometimes they are irregular or of the Cheyne-Stokes type, and they usually become slow before ceasing altogether. The dyspnœa is attended with great distress, and often with pain in the chest, referred to the seat of the embolism. No characteristic physical signs are to be made out. The face is sometimes pallid, sometimes cyanosed. The heart's action is tumultuous, and the pulse is rapid, weak, and irregular. The temperature falls and the extremities become cold and clammy. Although embolism of the pulmonary artery is nearly always fatal, recovery in rare instances has taken place. Sometimes the patient has several attacks before the fatal termination.

When a small or medium-sized branch of the pulmonary artery becomes blocked by embolism or thrombosis, the symptoms thereby produced are usually much less serious. Embolism of a medium-sized branch may be followed by symptoms such as dyspnœa and palpitation, similar to but less intense than those already described. Sometimes pulmonary infarcts are formed without giving rise to any special symptoms. The most characteristic symptoms are the expectoration of blood-stained sputa, and pain in the side. In cases of chronic congestion due to mitral stenosis or other forms of heart disease there may be copious hæmoptysis, but more commonly the sputa are scanty and of a rusty colour. Pain in the side arises if there be involvement of the pleura, and is accompanied with tenderness on deep pressure, and sometimes with pleuritic friction. There may be a rise of temperature with or without a rigor. When the embolism is septic, rigors are constant and symptoms of septicæmia with signs of pneumonia or abscess supervene.

Diagnosis.—The symptoms of embolism of a large branch, as described, are usually sufficiently characteristic; and when they occur in a case in which thrombosis in the veins of the extremities or in those of the uterus is known to exist, a diagnosis may safely be made.

Treatment.—Treatment can do little to avert death in pulmonary embolism when a large vessel is suddenly blocked. The pain and distress may be alleviated by hypodermic injection of morphine. It is rational, when dyspnœa continues, to perform

venesection, and so relieve the pressure in the venous system. Ether and ammonia or alcohol may be given in small doses at short intervals to help to maintain the heart's action until collateral pulmonary circulation is established.

BRONCHO-PNEUMONIA

Broncho-pneumonia is a disease which has received a variety of names, such as "catarrhal," "lobular," "vesicular," and "disseminated" pneumonia.

The anatomical changes have been described at p. 64, and the various causal relations have been considered in the section on general etiology. Clinically two forms may be recognised, the one primary, the other secondary. While the anatomical changes of these are indistinguishable, the primary form has been found to be more commonly associated with the presence of pneumococci, the secondary with streptococci.

Symptoms.—The primary form is principally met with in young children, in whom it runs a course similar to acute lobar pneumonia. The illness comes on suddenly without previous ill-health, and the main symptoms and the physical signs do not differ from those described below as characterising the secondary form. The temperature, however, is persistently high and only slightly remittent, and in favourable cases the pyrexia, after lasting six or seven days, terminates by crisis. Convalescence then sets in, and there is no tendency to collapse.

In the description given of capillary bronchitis (p. 161) it has been pointed out that in young children it is usually complicated with broncho-pneumonia. Some authorities make no distinction between the two conditions, which certainly have much in common, and the term "congestive broncho-pneumonia" has been applied to capillary bronchitis, but the symptoms of the latter are more intense and severe than those of ordinary broncho-pneumonia.

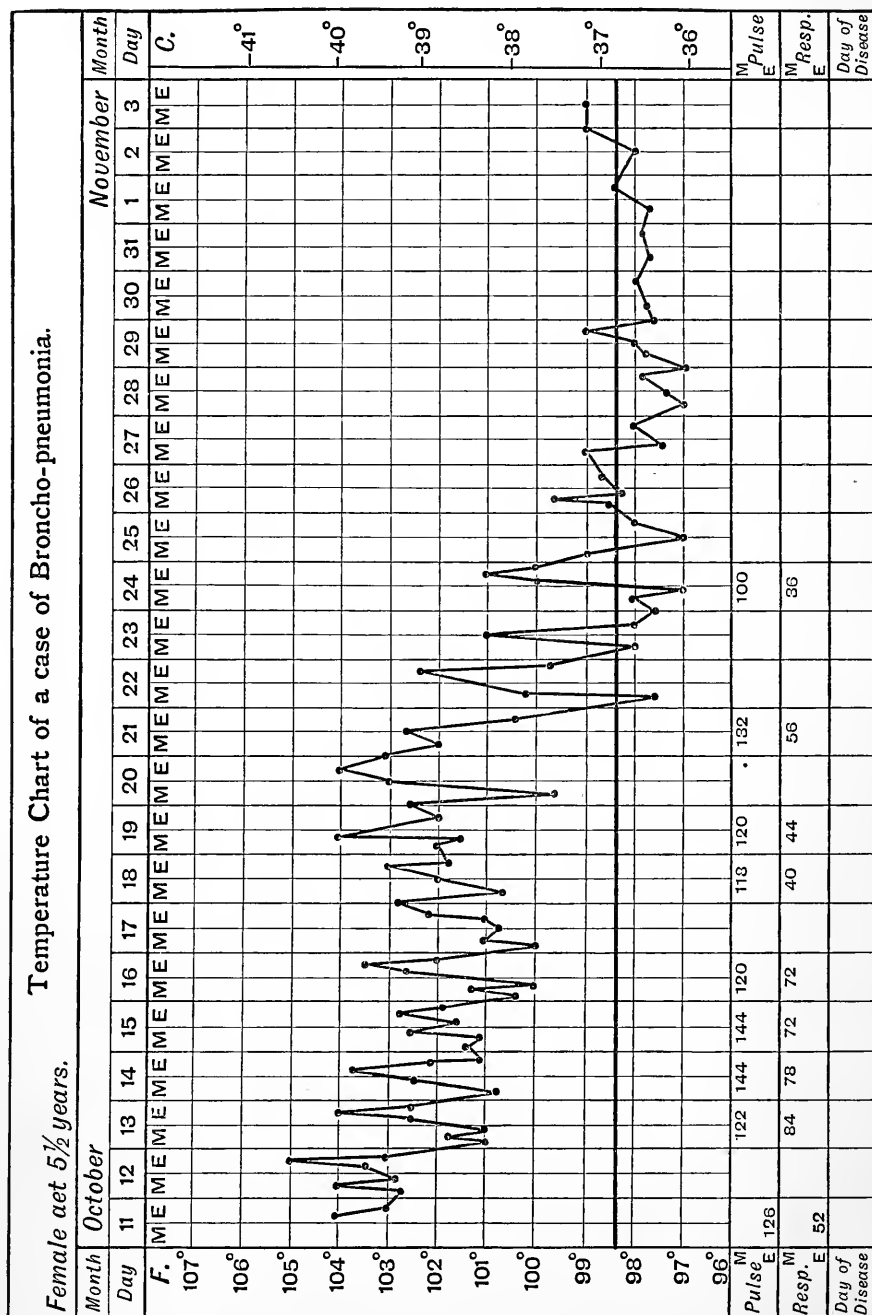
An acute primary form is occasionally but very rarely observed in adults. The illness sets in with the symptoms of acute bronchitis, and there is no pronounced rigor. The patient suffers from cough, dyspnoea, and pain in the side, usually accompanied with a considerable amount of prostration. The expectoration is mucous or mucopurulent, but not rusty-coloured. There is an irregularly remittent type of pyrexia which terminates by lysis, not by crisis, after a

period which varies in different cases from four or five days to two or three weeks. The percussion note is impaired and crepitant râles are present over the affected area, which is usually at one base, and rhonchi are audible on both sides. With convalescence the abnormal physical signs disappear and the patient makes a good recovery.

The secondary affection follows on acute bronchitis, measles, diphtheria, whooping-cough, or one of the other infective fevers, or is the result of aspiration of food or other irritating matter. As a sequel of bronchitis, it is seldom met with except in the case of young children and old people. Its invasion is gradual, with dyspnœa and fever, and is rarely marked by special symptoms, such as rigor, pain in the side, vomiting, or convulsions, which usher in acute lobar pneumonia. When the illness is fully developed the respirations are greatly accelerated, and there is marked dyspnœa, which may be accompanied with much distress. The thoracic movements are shallow and chiefly up and down, while in young subjects there may be considerable retraction of the lower part of the chest with each inspiration. The inspiration is relatively short, while the expiration is sometimes attended with a grunting sound. Cough is usually present, and is short, dry, and sometimes painful, little expectoration being brought up except after vomiting. The sputa, when there are any, are mucous or muco-purulent and tenacious, but very rarely rusty-coloured. The physical signs are those of bronchitis, combined with patches of consolidation and collapse. Harsh breath sounds with rhonchi and crepitations are generally audible. The crepitations usually accompany expiration and do not remain fixed in the same spots. Dulness and feeble or bronchial breathing are probably to be found at one or both bases, or near the vertebral column, or there may be patches of dulness and bronchial breathing in other situations. The signs of consolidation are seldom extensive, and clear up in one place to appear in another.

Fever is usually protracted in its duration, lasting two or three weeks or more, and is erratic in its course, marked remissions and exacerbations occurring daily but irregularly, as shown in the annexed chart. In young children the temperatures generally run high. Defervescence is gradual and usually extends over four or five days, while it may be interrupted by irregular exacerbations.

The pulse is rapid and may be extremely so in severe cases, when it is also small and feeble. Cyanosis becomes marked as the disease advances. Perspirations are not uncommon, and may be



general or limited to the head. There is loss of appetite and the lips and tongue become parched and dry, and aphthæ sometimes appear in the mouth. Vomiting is rare, but diarrhœa is not

unusual. Restlessness is often present, and there may be delirium at night, while as fever continues there is progressive wasting and loss of strength. In unfavourable cases the patient becomes drowsy and passes into a semi-comatose condition, and in some cases cerebral symptoms closely resembling meningitis supervene.

With the decline of the fever the other symptoms gradually disappear. Cough and the physical signs of bronchitis and consolidation may remain a long time.

Convalescence is usually tedious and apt to be interrupted by recrudescences.

Diagnosis.—Broncho-pneumonia is most commonly mistaken for bronchitis, acute pulmonary tuberculosis, or lobar pneumonia. The most important points are the presence of fever of the character described, and its association with dyspnoea and the physical signs which have been mentioned. Broncho-pneumonia may be overlooked in measles or other specific fever, and should be suspected when pyrexia continues beyond the usual time. In uncomplicated bronchitis, pyrexia is much less marked than in broncho-pneumonia. The symptoms of capillary bronchitis only differ from those of broncho-pneumonia in degree, and it is not possible to draw any hard and fast line of distinction between the two conditions. It is not an easy matter to distinguish collapse from broncho-pneumonic consolidation, but it may be noted that with collapse there is more retraction of the lower ribs. There is often great difficulty in differentiating broncho-pneumonia from acute pulmonary tuberculosis. In the former there is more rapid development of the signs of consolidation, which in the latter do not clear up when they have once appeared. In tuberculosis, prostration does not appear so early or so quickly as in broncho-pneumonia. The mode of onset and the longer duration of the fever, and its more marked remissions and exacerbations, help to distinguish the secondary form of the disease from acute lobar pneumonia, while in the primary form the diagnosis must rest chiefly on the physical signs, which point to consolidation, usually in patches on both sides, instead of more or less extensively over one lung.

Prognosis.—The prognosis is always grave, and is more so the younger the patient. The malady is very fatal in infants under a year old. A most dangerous form is that complicating whooping-cough, and the worst subjects are puny, ill-nourished, rickety infants. Among the unfavourable signs are much prostration, diminished power of cough and expectoration, extensive signs of collapse, temperature ranging over 105° F., great rapidity and weakness of

the pulse, somnolence, coma, and convulsions. The aspiration forms of broncho-pneumonia are usually fatal.

Treatment.—The serious nature of broncho-pneumonia makes its treatment a matter of great moment. The principal indications are to maintain the patient's strength and to relieve dyspnœa. It is usually recommended that the temperature of the room should be maintained at about 65° F., and that the air round the patient should be made moist by means of steam from a kettle or special apparatus. It is, however, of very great importance that the air of the sick-room should be pure, and it is found that cases do better when the air is freely admitted to the room than when the temperature is maintained by keeping the windows shut. The plan of surrounding the patient with curtains within which is directed a spray of steam is of doubtful advantage. The patient should be kept warm but not overburdened with bedclothes.

It is advisable to give an aperient early in the illness and to repeat it when the bowels are confined. Attention should be paid to the hygiene of the mouth and nose. If the tongue be furred, calomel is the best aperient. The food should be liquid and such as is adapted to the age of the patient. When there is prostration, alcoholic stimulants are of great value. In the case of an infant ten drops of brandy may be given to begin with every two hours.

A mixture such as the following may be appropriately prescribed for an infant:—

R Liq. ammon. citrat. ʒvjss.
Pot. citrat. gr. xxiv.
Oxymel. scill.
Syrup. limon. āā ʒj.
Aquam ad ʒij.

Ft. mist. Sig.—A teaspoonful to be taken every four hours

When expectoration is difficult, carbonate of ammonia and ipecacuanha may be added. If dyspnœa be urgent, emetics are valuable, and ipecacuanha may be given in doses sufficient to produce vomiting, and along with it the emptying of the bronchial tubes. The internal administration of belladonna has appeared to benefit, and the remedy is certainly very well borne by young children. Small doses of opium in the form of Dover's powder (gr. $\frac{1}{2}$ -1) may be cautiously given when the cough is frequent and ineffectual and there is much restlessness.

The chest may be rubbed with stimulating liniments, such as camphor or ammonia liniments, after which a jacket of cotton-

wool may be applied. Mustard and linseed poultices, lightly made and frequently changed, often relieve the breathing. The body should be sponged twice a day with warm or tepid water. Cold baths and cold applications to the chest have been largely employed, especially in Germany, in cases where dyspnœa is great, the temperature is high, and cyanosis is marked. The bath should be at a temperature of 65° to 67° F., and the immersion from one to three minutes. Cold compresses and cold sponging are equally effectual and simpler. Whichever method be used the pulse and respiration should be closely watched, and if the cold application be doing good the respirations should become slower and deeper, the pulse quieter, and the temperature lower.

In cases of marked cyanosis, oxygen inhalations may be employed, the gas being warmed by passing it through warm water, as elsewhere recommended.

PULMONARY COLLAPSE

The causation and anatomical changes and appearances of congenital and acquired collapse have been considered and described at pp. 85-87.

Symptoms.—The condition of the infant with unexpanded lungs, if it survive its birth, is characterised by great feebleness and low temperature. Respiration is very superficial, and there are probably paroxysmal attacks of dyspnœa. There is pallor combined with cyanosis, which increases in intensity. The infant is somnolent and disinclined or unable to suck, and its cry is feeble. Carpo-pedal contractions and slight convulsive movements are not infrequent. On examination the signs are very imperfect expansion of the chest and sinking in of the intercostal spaces, together with defective inspiratory murmur after the feeble cry, but percussion may be little if at all impaired. Life, as a rule, is only maintained for a few days, but in exceptional cases the infant has survived for some weeks.

The symptoms and physical signs of acquired collapse must be considered along with the conditions from which it arises. The slighter degrees of collapse give rise to neither symptoms nor physical signs which are characteristic. When extensive areas of the lung are collapsed in the course of bronchitis or whooping-cough, there is usually much dyspnœa with cyanosis and prostration. There is inspiratory retraction on one or both sides, especially at

the base. Over the affected areas the breath sounds may be of a bronchial character, but more usually they are diminished or are altogether inaudible, and the percussion note is dull or impaired.

Treatment.—In atelectasis of the new-born, care must first be taken that the upper air-passages are free. Attempts may be made to excite deep breathing by immersing the infant in a warm bath and then dashing cold water on the chest, or by slapping the nates. The child should be kept warm. Artificial respiration may be tried from time to time.

ACUTE PNEUMONIA

Acute pneumonia is variously known as “lobar,” “croupous,” or “fibrinous” pneumonia, “pleuro-pneumonia,” “pneumonitis,” “pneumonic fever,” and “lung fever.” In some text-books it is included among fevers or general diseases, but there is obvious convenience in considering it among diseases of the respiratory system. Its relations to micro-organisms have been discussed at p. 56, and the other causal factors at pp. 50-56. The morbid anatomy is described at pp. 65-69. An account of “epidemic pneumonia” has been given in Vol. I. pp. 178-181.

Symptoms.—As a rule, the illness begins suddenly with a rigor and rise of temperature, but the onset is sometimes preceded by bronchial catarrh or general malaise for one or two days. The rigor, which is usually severe, seldom lasts longer than from ten to thirty minutes, and is rarely repeated, while the rise of temperature is not at once followed by any marked fall. Vomiting sometimes replaces the rigor, particularly in the young and the aged, and in children the illness may be ushered in with convulsions, headache, and delirium.

The main symptoms of the acute stage are fever, pain in the side, short, frequent, and hacking cough with thick, tenacious, and often rusty-coloured expectoration, increased rapidity of respiration and pulse, dyspnoea, prostration, heat and dryness of skin, thirst, loss of appetite, constipation or looseness of the bowels, headache, and not infrequently mental disturbance. The physical signs of consolidation of the lung soon make their appearance. For from three to ten days the patient gradually becomes more and more ill, and then in favourable cases there is a very decided and rapid change for the better; the fever subsides, the respiration and pulse slow down, dyspnoea and distress pass away, free perspiration

breaks out, and the mind, if previously affected, becomes clear. During the stage of convalescence, which thus commences, like the illness itself, somewhat abruptly, strength is usually quickly regained and the physical signs of pulmonary consolidation soon, as a rule, completely disappear.

We shall now proceed to consider the symptoms in detail.

Pyrexia.—A rapid rise of temperature at the onset and continuance of fever with only slight remissions, the temperature being usually slightly higher in the evening than in the morning, are characteristic. Sometimes, however, the initial rise is more gradual, and while the fever lasts sudden rises and falls may occur irregularly. High temperatures are not uncommon, especially early in the illness and in the case of children.

The fever commonly terminates by crisis, in which case a fall of temperature to or below the normal may take place within the space of twelve or twenty-four hours (see Chart, p. 198). After the fall, sometimes the temperature does not again rise above normal, but occasionally there are temporary rises, though rarely to the extent of more than two or three degrees. The crisis occurs comparatively frequently on the seventh day, but may take place earlier or later at any time between the second and eighteenth days. Fever is usually continued beyond the average period in apical cases. In exceptional cases defervescence is gradual or the crisis is incomplete and fever continues, or three or four days after the crisis a relapse occurs which is attended with a return of symptoms and signs of a fresh pulmonary invasion. When the temperature, after falling at the crisis and remaining nearly normal for some days, then slowly rises again and assumes a remittent character, empyema should be suspected.

Pain in the side is one of the most constant as well as one of the earliest symptoms, and is usually very acute. It is due to the affection of the pleura, and is aggravated by taking a deep breath and by cough, and is usually accompanied by marked tenderness on deep pressure over the affected part. It is important, however, to bear in mind that sometimes the pain may be felt in the belly or the back, so that its situation may mislead as to the nature and seat of the disease. This results from the intercostal trunks being involved (see p. 118). The pain generally subsides after a few days, but sometimes it lasts to the end of the acute stage of the illness.

Respiratory symptoms.—The respirations are quick and shallow and usually number from 35 to 40 per minute, but may reach 60,

short and hacking cough is an early and almost constant symptom in young adults, although it may be absent in the old. The sputa may be frothy at first, but soon become viscous, and usually have a reddish-brown, saffron, or typical rusty colour. Sometimes, however, they are more watery and of a dark purple colour resembling prune juice; sometimes bright red like blood, and sometimes greenish. Occasionally they are mucous, as in bronchitis, but more viscid and tenacious. Sometimes, especially in the case of children and old people, there is no expectoration, and both cough and expectoration usually cease before a fatal termination. On examination by the microscope the sputa are found to contain pneumococci, blood-corpuscles, epithelial cells, and fibrinous casts of the bronchioles and alveoli.

Physical signs.—In the early stage fine crackling râles are usually to be heard over the affected area, while the breath sounds are weaker or harsher than normal. Crepitation may continue or disappear during the next stage, when the characteristic signs are those of consolidation—dulness, bronchial or exceptionally suppressed breath sounds, bronchophony, pectoriloquy, increased vocal fremitus, and diminished movement. During the stage of resolution there is a return of crepitation, should it have disappeared, which *crepitation redux*, as it has been called, is generally of a coarse quality, while there is a gradual disappearance of the signs of consolidation, of which dulness on percussion is, as a rule, the most persistent. Resolution usually takes place more rapidly in children than in adults; sometimes abnormal physical signs remain for only a few days after the crisis, but more commonly they do not disappear for a week or a fortnight.

Circulatory symptoms.—The pulse is full and rapid, although exceptionally in old people it may be infrequent. Marked quickness with low tension of pulse, such as would probably characterise a rate of 120 or over in an adult, is a sign of prostration, and is unfavourable, but in the case of young children the pulse may be very rapid, 160 to 180, without there being any special danger. A feeble pulse and a feeble first sound at the apex point to failing heart. The colour of the lips is a measure of the impediment to the circulation, and any marked degree of cyanosis is an unfavourable symptom.

The blood.—There is a marked increase in the amount of fibrin, the proportion of which rises from two or three parts per thousand to between four and ten parts per thousand; and this is probably an important factor in the production of thrombosis, which may

occur in the pulmonary artery during the acute stage or in the peripheral veins during convalescence. Leucocytosis occurs very early in the disease and continues throughout the febrile period. It is of the active polymorphous variety, the actively amoeboid corpuscles being increased in greater proportion than the other forms, while the eosinophile cells are usually greatly reduced in number. Absence of leucocytosis is a bad sign, except in mild cases, but the reverse, that its presence is a good sign, does not hold true. Leucocytosis gradually disappears after the crisis, and its persistence points as a rule to the presence of some complication. Pneumococci are probably always present in the blood during the febrile stage, but only in severe cases are they in sufficient number for even a skilled investigator to be able to find them.

The skin.—The face is usually flushed, especially over the malar bones, and sometimes the flush is limited to one cheek, commonly on the same side as the affected lung. The skin is pungently hot and, as a rule, dry. Sweating to any marked extent during the febrile stage is generally a bad sign, and occurs chiefly in drunkards and in cases of great prostration. At the crisis, however, profuse perspiration is almost constant. Herpes not infrequently breaks out on the face, usually about the lips or the alæ nasi, but sometimes on the cheeks or ears. It is seldom seen before the third or fourth day, and it may come out in successive crops. It may be delayed until the crisis, but its appearance during the febrile stage is regarded as, on the whole, favourable. Other skin eruptions are not common. Miliaria, sudamina, acne, urticaria, and boils are sometimes observed, and multiple cutaneous gangrene, ecchymoses, and purpura have been met with in severe cases.

Digestive system.—There is usually more or less complete anorexia and decided thirst. The tongue may be clean, but more commonly is thickly coated with a creamy fur and, in severe cases, is dry and brown. Vomiting, as has been mentioned, may be an early symptom, especially in the case of children, and it may be repeated during the course of the illness. Diarrhœa, while less common, may occur at any stage, but is more frequent at the beginning or at the crisis. The combination of vomiting and abdominal pain has more than once led to the erroneous diagnosis of appendicitis, peritonitis, or intestinal obstruction, and laparotomy has actually been performed through this mistake. The spleen in some cases is enlarged and palpable, especially in epidemic pneumonia.

The urine.—During the febrile stage the urine is decreased in amount and increased in acidity and density, being usually loaded with urea and containing an excess of uric acid, while there is a marked diminution of the chlorides. Albuminuria is very frequent, especially at the height of the illness; and the amount of albumen, while usually slight, is to some extent a measure of the intensity of the disease. Casts and blood are sometimes temporarily present. In cases complicated with chronic renal disease the specific gravity may remain low and the excretion of urea show no increase. After the crisis the chlorides reappear in large amount, the excretion of urea diminishes, the albuminuria disappears, and the reaction, previously strongly acid, becomes for a day or two alkaline or neutral.

Nervous system.—During the first few days headache is frequent and may be severe. Delirium in a mild form, characterised by wandering and mental confusion, is common, but it may assume a grave form, so that sometimes the case has been taken for one of acute mania. In drunkards not infrequently the symptoms of delirium tremens, including sleeplessness, talkativeness with incoherency, illusions, and tremors, may precede by several days the appearance of the physical signs of pneumonia. In some cases, especially in elderly subjects, the delirium is of a low, muttering type and is attended with profound prostration. Sometimes delirium continues after the crisis; sometimes its onset is delayed until about the time of the crisis; and in some cases the crisis is followed by acute mania or dementia. The average duration of the post-pyrexial delirium is ten days. Delirium, except in a mild form, is a sign of danger, and its intensity is proportional to the degree of toxæmia. Coma may succeed delirium or may come on independently, especially in children and old people.

Convulsions are rare in adults but not uncommon in children, in whom there may also be severe headache, delirium, strabismus, and prostration. The illness thus sometimes resembles, and has been mistaken for, tuberculous meningitis, some of the reported recoveries from which are no doubt to be explained in this way.

The pupils in pneumonia are generally somewhat dilated. Some observers have noted that they are frequently unequal, especially in apical cases, the larger pupil being on the side of the lesion. It has been suggested that, like the unilateral flushing, this is a sympathetic phenomenon, but its existence has been questioned by good authorities.

Modes of termination.—*Recovery* has already been referred to as the most usual mode of termination.

Relapses occasionally occur, but are decidedly rare. They seldom come on more than a few days after the crisis, though sometimes the interval is seven or eight days. They are attended with a return of all the symptoms and the physical signs of a fresh invasion of the lung. The fever is, however, as a rule of a shorter duration than in the primary attack. Very exceptionally a second and even third relapse occurs.

Delayed resolution.—While in the majority of cases the physical signs of consolidation soon disappear, in a few cases resolution is delayed and the signs of consolidation persist. Most of the cases of delayed resolution ultimately end in recovery, although in some cases chronic induration may ensue (see Chronic Pneumonia, p. 212).

Rarely acute pneumonia is followed by *abscess of the lung* or by *gangrene*, both of which conditions will be subsequently considered.

Death.—Unfortunately, in a large proportion of cases (one out of five in hospital practice) the disease proves fatal, as a rule in the acute stage of the illness. When the illness is going to terminate in death, usually the prostration becomes more intense or the dyspnoea more extreme. The pulse becomes feebler and more rapid; the respirations become shorter, shallower, and quicker. Expectoration becomes more difficult or may cease altogether, while coarse râles and rhonchi are generally audible all over the lungs. The face becomes livid and the extremities cold, while profuse perspiration breaks out over the body. The mind may remain clear almost to the end, or delirium may be followed by a semi-comatose condition. In children convulsions or coma may precede death, while in old people the end may be sudden. Death may also occur during the crisis from collapse, or it may ensue at a later period from the effects of sequelæ or complication.

Complications.—*Laryngitis* is a rare and usually unimportant complication, but œdema of the glottis has sometimes directly contributed to a fatal issue. *Bronchitis* is more frequent, and when diffused or associated with pre-existing emphysema is serious, aggravating the dyspnoea and cough and increasing the amount of expectoration. *Pleurisy* is an almost constant accompaniment, but, as a rule, the amount of effusion is small. It only becomes of importance when the effusion increases as it sometimes does after the crisis, when it occurs on the opposite side to the pneumonia, and in particular when it is purulent. *Empyema* is a not very infrequent sequel to pneumonia, and should always be thought of when, after the crisis, pyrexia continues or returns. An empyema

after pneumonia is not infrequently loculated, and sometimes even with careful physical examination it may be difficult to exactly locate it. When there is good reason to suspect it the exploring needle should be employed.

Pericarditis is less common, but much more serious than pleurisy. It adds greatly to the gravity, about 50 per cent of the cases in which it occurs proving fatal. It is more frequent but less dangerous in young subjects than in adults. *Acute endocarditis* is not common except when there is pre-existing chronic valvular disease, in which case it sometimes assumes the ulcerative form. *Thrombosis of the pulmonary artery* is often a terminal process in fatal cases. During convalescence, thrombosis may occur in the veins of the lower extremities, or less commonly in those of the upper extremities.

Parotitis is a rare complication, and is usually of bad augury. It may result in diffuse suppuration of the tissues of the neck or terminate in gangrene. *Vomiting* may sometimes prove troublesome, while severe *diarrhœa* may be produced by a croupous form of colitis. A slight degree of *icterus*, the cause of which is obscure, is not uncommon. It may be associated with nausea and vomiting, but seldom lasts longer than a week.

Among other complications which may be mentioned are nephritis, peripheral neuritis, meningitis, and acute synovitis. *Acute nephritis* is rare, but chronic renal disease, when an associated condition, adds greatly to the gravity of the illness. *Peripheral neuritis* is decidedly uncommon, and probably results from some other disease, such as influenza, with which pneumonia has been associated. *Meningitis* is an infrequent but interesting complication, which may arise from a pneumococcic infection of the membranes. *Acute arthritis*, usually of a suppurative type affecting one or more joints, has been recorded in a few cases, and this also is a manifestation of pneumococcic infection. As a rule it follows soon after the crisis, but may precede or accompany the lung affection. The joints of the upper extremities, especially the sterno-clavicular articulations and the shoulders, the knee-joints, and joints the seat of old injury are most likely to be affected.

VARIETIES OF PNEUMONIA.—A considerable number of varieties of pneumonia have received separate description. While on the whole the clinical features of the *epidemic form* of pneumonia do not differ from those of the sporadic or ordinary form, it has been observed that in some epidemics cerebral symptoms, in some cardiac symptoms, and in others gastro-intestinal symptoms predominate. Cases of *apical pneumonia*, as a rule, are of an adynamic

type and have marked cerebral symptoms, while the pyrexial stage is of more than average duration. Cases are met with in which, while the symptoms and course of the disease are typical, abnormal physical signs are often very late in appearing or are absent altogether. It is probable that these are cases of *deep-seated pneumonia*. Other cases are of an *abortive type*, the symptoms being typical until the second or third day, when the fever ceases abruptly. Pneumonia is frequently *secondary* to other diseases, in which case both the symptoms and the course are atypical, being modified by the primary disease. Of other varieties we need only mention the latent type, the typhoid or asthenic type, the migratory or wandering type, and influenzal pneumonia.

To the *latent type* belong certain cases in children, in old people, and in drunkards where the cerebral or abdominal symptoms predominate and mask the true nature of the case.

To the *typhoid or asthenic type* belong those cases where the invasion is gradual instead of sudden, and where headache, stupor, and low muttering delirium are the most marked symptoms. The pneumonia is not infrequently multiple, affecting both lungs or involving the apices. The sputa are sanguineous or of the "prune juice" type, or offensive and purulent. If recovery follows, it is only after a protracted course, and resolution is slow. Irregular types of this kind are perhaps most common in elderly subjects.

Migratory or wandering pneumonia.—This form of pneumonia is probably due to infection by the streptococcus pyogenes. The onset is often less brusque than that of ordinary pneumonia, but the same symptoms, malaise, feverish and chilly sensations, pain in the side, and cough, are usually present. The sputum is more purulent and less often rusty, and contains streptococci as well as pneumococci. The fever is irregular and may be of a relapsing type, accompanied with rigors and exacerbations, with affection of new parts of the lung. There is no crisis, and the fever subsides by lysis after lasting sometimes for three or four weeks. The physical signs are usually late in appearing, there is a marked tendency for the local process to wander, and the upper lobes are more frequently involved than is the case in ordinary pneumonia. The cough is apt to be troublesome as long as abnormal physical signs remain. Convalescence is likely to be long, fatiguing, and attended with great prostration, so that it may be months before the patient has recovered from the effects of the disease. The differential diagnosis of this special type must be made from the abnormal features of the case and the discovery in the sputum of streptococci.

Influenzal pneumonia.—It is maintained by some authorities that the pneumonia of influenza results from streptococcic infection, and Finkler found this microbe present in every case of the kind where micro-organisms could be demonstrated. The clinical history in some cases resembles that of the streptococcic pneumonia just given. The onset may be sudden with sharp rise of temperature, but there is no marked pain or rusty sputum. Abnormal physical signs may not show themselves for three or four days, and then there may be several areas of consolidation which vary in situation from day to day, abnormal signs disappearing in one position only to appear in another. Expectoration is sometimes profuse and purulent or mucopurulent, or entirely absent. The duration is much more protracted than that of ordinary pneumonia. In other cases the pneumonia has assumed what may be called a fulminant type, prostration being a marked feature from the first, and the illness ending fatally after a few days. Influenzal pneumonia has appeared to be contagious independently of influenza itself.

PNEUMONIA IN CHILDREN.—Vomiting, headache, and pain in the belly are the most constant early symptoms. Pain in the belly is more common than pain in the chest, and it may be associated with constipation or diarrhoea. There is usually a high degree of fever. The headache is often combined with delirium or drowsiness, but it is characteristic that after a time it passes off, while in meningitis or cerebral abscess it persists. Cough is generally unattended with expectoration, and is frequently absent altogether, and the appearance of abnormal physical signs may be delayed. The increased rapidity of respiration, and the altered pulse-respiration ratio, greatly assist in forming a correct diagnosis. Expiration is often accompanied with a grunting noise. Pneumonia in children usually runs a favourable course.

PNEUMONIA IN OLD PEOPLE, as a rule, comes on insidiously, without a decided rigor, although there may be a feeling of chilliness. The patient first complains of feeling tired and weak, and prostration may supervene quite early. The appetite is soon lost, so that there is great difficulty in getting the patient to take food, and the tongue becomes dry and brown. The face is pallid rather than flushed, and the eyes are sunken. Pain in the side may be absent, while pains may be felt in the head, back, or limbs. Dyspnoea and cough may be little marked, and expectoration may be absent, or transparent, viscous, or simply puriform, and free from rusty tint. The temperature probably does not rise above 102° F. The heart shows signs of failing power, and the pulse is feeble, irregular, and

probably intermittent. Physical signs of consolidation are not well marked, and are usually limited to a small area. Pneumonia in old people is always a very grave affection, and the proportion of fatal cases is very high.

Diagnosis.—In the majority of cases the diagnosis of pneumonia is no difficult matter. Mistakes most frequently arise in the case of children, in old people, in alcoholic subjects, and in persons in whom pneumonia is superadded to some pre-existing disease.

Sometimes the head symptoms so greatly predominate, especially in the case of children, that the disease may be mistaken for meningitis or cerebral abscess. Both in children and adults, where abdominal symptoms are prominent, pneumonia may simulate appendicitis, peritonitis, or obstruction. The high initial temperature and its subsequent slight variations, and the altered pulse-respiration ratio, are important points of distinction before definite physical signs of pneumonia have appeared.

In old people the symptoms may not be well marked, but the altered respiration rate should attract notice to the lungs. In alcoholic subjects, especially when delirium comes on early, the disease may be taken to be delirium tremens, an error which generally has only to be remembered to be avoided.

Pneumonia has also been mistaken for acute mania, typhus fever, or enteric fever. In all these cases a careful physical examination of the chest will usually lead to a correct diagnosis. In pleurisy with effusion the onset is more gradual and the symptoms are usually quite different from those of pneumonia, there being less fever, less constitutional disturbance, and less acceleration of the respiration. Cases of pneumococcic pleurisy have, however, been recorded, in which the symptoms closely resembled those of pneumonia. The physical signs of effusion will be found instead of those of consolidation.

Prognosis.—After childhood the danger from pneumonia increases steadily with age. The mortality is least in children between five and fifteen, and greatest in infancy and in old age. Females show a slightly higher rate of mortality than males, and the influence of pregnancy is unfavourable. The mortality is high among those debilitated by privation, intemperance, or unhealthy occupation or mode of life, among persons constitutionally weak or of poor physique, and among those affected with emphysema or cardiac or renal disease. The greater the extent of lung involved the greater the danger, and pneumonia of the apex is more serious than a basic affection. Great rapidity of the respiration and marked

dyspnœa or orthopnœa are unfavourable. It is a bad sign when the sputa are thin and have the appearance of prune juice or liquorice juice, or are diffuent and puriform, or when there is profuse hæmoptysis.

Persistently high temperature, or a pulse rate over 120, except in young subjects, gives cause for anxiety, and it is a bad sign when the pulse is feeble, dicrotic, and of low tension. Pericarditis is a complication which greatly increases the danger. Great prostration, the low muttering form of delirium, and violent delirium coming on late in the illness are all of bad omen.

Treatment.—There is at present no specific treatment. Encouraged by the success which has attended the serum treatment of diphtheria, Dr. Washbourn in this country and G. and F. Klemperer, Pane, and others abroad have tried a serum treatment for pneumonia. The serum used on a large scale has been obtained from horses and donkeys rendered immune by injections, first, of pneumococcus cultivations, the virulence of which has been destroyed by heat, and, second, of successively larger quantities of living cultivations. This serum is antibacterial but not antitoxic, and the results so far have been inconclusive as to its value. We have no means of cutting short the fever or of directly attacking the cause of the disease, or of checking the advance of the morbid process in the lung. Our efforts, therefore, are directed to tide the patient through the period of danger, to maintain his strength, promote his comfort, and alleviate his sufferings.

The patient should occupy an airy, clean, cool, and well-ventilated room. Fresh air is of the utmost importance, and, if possible, the temperature should not be above 60° F. It is hardly necessary to say he should be kept in bed, and care should be taken that this is comfortable, and that the blankets, while sufficient, are not too heavy.

The most suitable diet consists of milk, eggs, beef-tea, Benger's food, rusks, and calf's-foot jelly. The mouth and teeth should be kept thoroughly clean. At the outset it is well to secure a good evacuation of the bowels, and 2 or 3 grains of calomel may be given at bedtime, followed by a saline aperient next morning, aperients being repeated later on if necessary. If there be diarrhœa, small doses of calomel combined with Dover's powder prove useful. In ordinary cases one may give a neutral effervescing saline mixture, or, in the case of children, the solution of citrate or acetate of ammonia.

For the relief of pain in the side, linseed or mustard and linseed

poultices, hot fomentations, hot-water bottles, turpentine stupes, belladonna or belladonna and chloroform liniment, leeches, or an ice-bag may be applied. If the pain be severe, and be unrelieved by local applications, a hypodermic injection of morphine may be administered. For the relief of cough, simple remedies, such as warm milk, barley-water or linseed-tea, liquorice lozenges, menthol pastilles, or a plain linctus, are the best. Expectorants, as a rule, do not benefit. When the temperature is high, tepid sponging, cold compresses, the cold pack, the application of an ice-bag, or ice cradling may be tried. For sleeplessness, chloralamide, sulphonal, and paraldehyde are useful drugs, and tepid sponging helps to promote sleep. Opium and alcohol, however, are of great service when used with discrimination. In the case of delirium, ice compresses to the head, the cold pack, saline infusion or saline subcutaneous injection, and alcohol are the most useful remedies.

As to the use of opium in pneumonia, widely different opinions are held even at the present time, some regarding it as most beneficial under certain conditions, others as dangerous or to be employed only with the utmost caution. We believe, however, that it may be given with advantage when there is severe pain, troublesome harassing cough, or sleeplessness or restlessness.

Alcohol is also a remedy of great value, but one not to be employed simply as a matter of routine. The indications for its use are marked prostration, feebleness, irregularity, or great rapidity of the pulse, extreme rapidity of respiration, profuse sweating, tremors, muttering delirium, or delirium or collapse coming on at the crisis. The amount to be given under such circumstances will vary in individual cases, but from 4 to 8 ounces of brandy may be considered as much as is generally necessary in the twenty-four hours.

In cases of failing heart, in addition to alcohol, other cardiac stimulants, strychnine, caffeine, and ammonia, may be given.

Venesection is a most valuable means of treatment in certain cases. It is indicated when there is marked dyspnoea, or cyanosis with small pulse, and in cases of rapid invasion of a large tract of lung.

Oxygen inhalations are of service for the relief of dyspnoea. The oxygen should be warmed by passing it through warm water, and a large funnel may be used with advantage instead of the ordinary nozzle. In very severe cases the oxygen should be given continuously if it ease the patient. In other cases it may be given at intervals for ten minutes at a time.

Saline infusion or subcutaneous injection should be employed

when there is much prostration or profound toxæmia. Great benefit results from its use in such conditions, and it may sometimes be combined with venesection with advantage.

At the crisis, treatment should be directed to counteracting any dangerous depression, and should this arise stimulants may be given internally and heat applied externally. During convalescence the best remedies are fresh air and good food, but cinchona bark and acid are useful adjuvants.

PULMONARY ABSCESS

The pathology of pulmonary abscess has been considered at p. 72.

Symptoms.—There is probably a history of preceding pneumonia or of one of the other causes of the malady. At first there may be only fever of a hectic type, with perhaps occasional rigors. After a time, in some cases, a large amount of pus is suddenly expectorated, after which the signs of a cavity may be discovered, and the pyrexia temporarily declines. Before the evacuation of the abscess the physical signs are generally ill-defined, and it may be only after careful and repeated examination that there is discovered a patch of localised dulness, over which there is absence or diminution of breath sounds, together with, in some cases, local tenderness on firm pressure with the finger. Fœtor of the breath and sputa may or may not be present; this occurs and reaches a high degree of intensity when the formation of abscess is associated with gangrene, in which case the sputa and the contents of the abscess are usually of a brown colour, and consist partly of altered blood, with shreds of broken-down lung tissue. In other cases fœtor may be absent, at any rate for a time, and the sputa are abundant, puriform, and usually grass-green in colour, although possibly brownish at first, and contain masses of pigment mixed with tiny shreds of pulmonary tissue.

Prognosis.—The course of the illness varies. The gangrenous cases nearly all prove fatal, although the chances of recovery are slightly improved by operation. Of the other cases, after evacuation, spontaneously or by operation, some end in recovery, while others prove fatal from exhaustion, from septicæmia, or from successive attacks of pneumonia. The prognosis is worst in the most acute cases, in cases where there is affection of the opposite

lung, and in cases where the abscess contents are of a dark brown colour and have an extremely offensive odour.

Diagnosis.—Abscess of lung may be difficult to differentiate from a loculated empyema and from bronchiectasis. The character of the sputa and the presence in them of pulmonary tissue, and the nature of the physical signs, will help to distinguish from empyema. The shorter duration, the smaller amount of expectoration, and the position and nature of the physical signs, will help to exclude bronchiectasis.

Treatment.—If the abscess have evacuated itself through the lungs, the case may for a time be watched, the patient being placed under the best hygienic conditions. If decided improvement follow, then constitutional treatment may be continued. If, however, the abscess have not evacuated itself or if improvement have not followed evacuation, operative measures should be adopted. The suspected site of the abscess should be explored with the needle of an aspirator, preferably without an anæsthetic. When the abscess has been localised it should be opened up under chloroform, the patient lying on the back or on the affected side. A drainage tube long enough to reach the bottom of the cavity should be inserted and should be retained in place, except when removed for washing, until expectoration has ceased and the external discharge has almost disappeared.

PULMONARY GANGRENE

The causation and morbid anatomy of pulmonary gangrene have been discussed at pp. 71, 72.

Symptoms.—In some conditions such as embolism, or in insane patients, characteristic symptoms of gangrene may be altogether wanting or may be overshadowed by those of the primary disease. The illness may set in with rigors and pain in the side, with hæmoptysis, or with prostration and fever. Rigors are frequent and generally recur; pain in the side is not uncommonly very severe; hæmoptysis may be profuse and repeated; and the prostration is great. These symptoms are, in most cases, soon followed by others more specially typical, namely, fœtor of the breath and fœtor of the sputa, which are usually, but not always, present together. Fœtor of the breath may be a very early symptom, being specially noticeable after the patient coughs. The fœtor, both of the breath and sputa, is intense and intolerable.

Fœtor has been observed to be absent in the insane and in diabetic and embolic cases, and occasionally it is an intermittent phenomenon.

The sputa are always diffuent, more or less opaque, often of a dirty brown or chocolate colour, sometimes like pure blood and sometimes resembling the sputa of bronchiectasis, and of a dirty yellowish-green colour. They often contain fragments of pulmonary tissue, Dittrich's plugs, crystals of fatty acids, leucin and tyrosin, and numerous micro-organisms. The amount of the sputa is variable, being sometimes small and sometimes, as in cases secondary to bronchiectasis, very large. Cough is usually present, but is not troublesome except when expectoration is profuse, and then it may be paroxysmal. Dyspnœa may be considerable, especially in the acute pneumonic form.

The physical signs are at first those of consolidation, but later there may be signs of excavation.

Fever is usually present, the temperature, especially in the pneumonic cases, ranging high; but it may be very slight or absent. As a rule prostration becomes more and more marked. The pulse is small and rapid, and there is often profuse sweating. The patient generally lies on the back or on the affected side. The face is ashy pale, the tongue dry and brown. A condition of stupor or delirium usually supervenes before the fatal ending which terminates most cases.

Prognosis.—Death usually ensues rapidly between the fourth and fourteenth days, sometimes from collapse, sometimes from profuse hæmoptysis, pneumothorax, or hæmorrhage into the pleura, and sometimes from cerebral abscess, septicæmia, or pyæmia. Occasionally the illness is more chronic and lasts one or two months, and only in rare cases does it terminate in restoration to health.

Diagnosis.—The most important points are the existence of one of the causes of gangrene, the intense fœtor of the breath, the peculiar and horribly offensive sputa, and the amount of constitutional disturbance. Mistakes are most commonly made, on the one hand, in cases of fœtor associated with hæmoptysis, fœtid bronchitis, bronchiectasis, phthisis, pulmonary abscess, and perforating empyæma, and, on the other, in cases where characteristic symptoms are absent. When the disease is latent we cannot recognise it. In the other cases the resemblance is more superficial than real, and a correct diagnosis may usually be arrived at by a consideration of the points which have been mentioned.

Treatment.—Medicinal treatment as a rule is quite powerless to influence the progress of the disease and to avert the fatal issue. If there be signs of an abscess it should be dealt with surgically. An abundant supply of fresh air, with nourishing food and alcohol, may do something to improve the patient's condition. It will give the patient the best chance of recovery if he be kept out of doors in bed night and day, properly protected from cold and wet. The fœtor may be masked to some extent if the patient use an oronasal inhaler, on the sponge of which is dropped a creosote and menthol solution, or if dishes containing pine sawdust on which oil of turpentine is from time to time sprinkled be placed in the patient's vicinity. The internal remedies which have been employed for bronchiectasis may prove useful here, and apparent benefit has followed the administration of guaiacol (mv), oil of turpentine (mx), or myrtol (gr. ijss), in capsules three times a day. Guaiacol or guaiacol diluted with sterilised oil has also been given by subcutaneous injection.

CHRONIC PNEUMONIA

The conditions included under the head of chronic pneumonia are of somewhat varied origin and of different nature, as will be gathered from the account given at pp. 69-70 of the pathology and morbid anatomy. Many cases of chronic pneumonia are complicated sooner or later with bronchiectasis and present the symptoms of that disease. Others, as already pointed out, are cases of chronic pulmonary tuberculosis, where fibrosis and contraction of a limited portion of lung have occurred, and the tuberculous process has undergone arrest.

Symptoms.—Chronic consolidation of the lung very exceptionally is the result of an attack of acute lobar pneumonia. After the acute stage has passed away a slight degree of pyrexia persists. The patient continues to suffer from cough, which may be slight or troublesome, and he expectorates mucous or mucopurulent secretion. He remains short of breath, and pain in the side sometimes continues. At the same time it is found that the signs of consolidation do not disappear. Gradually the symptoms diminish, but cough with scanty expectoration and some dyspnoea on exertion persist. The affected part of the lung remains dull, and the chest movements are deficient and bronchial breathing is present over the affected area. At a later period symptoms of

bronchiectasis may supervene. The illness is usually of long duration.

In other cases which have been described as "subacute indurative pneumonia," or "primary parenchymatous pneumonia," the commencement of the illness is less acute than ordinary lobar pneumonia. At the onset there are pyrexia, dyspnœa, and cough, but the patient may not be confined to bed for more than a day or two. The pyrexia throughout is slight and irregularly remittent, and does not terminate by crisis. The sputum is seldom rusty, and is usually abundant. The physical signs of consolidation are of slower development, and persist when they have once appeared. The general condition of the patient gradually gets worse, and the sputa often become foetid. Some cases are complicated with gangrene. The illness is seldom prolonged for more than a few months.

In cases of chronic pneumonia which result from the inhalation of dust (pneumoconiosis) the prominent symptoms are cough and dyspnœa, and the illness closely resembles chronic bronchitis. In the case of miners and others who inhale dust consisting mostly of carbon particles the sputum is usually black, and is often large in amount. Occasionally it is blood-stained, and in exceptional cases profuse hæmoptysis occurs. Dyspnœa is generally an early symptom, and may be severe and urgent. The patient is usually pale and anæmic.

The physical signs are those of emphysema, often combined with induration and excavation. As time goes on the dyspnœa and cough become more troublesome and the amount of expectoration gradually increases.

Diagnosis.—The chief difficulty is in distinguishing chronic pneumonia from chronic pulmonary tuberculosis. One must be guided by the mode of onset and by the presence or absence of bacilli in the expectoration. Chronic pneumonia is, as a rule, a unilateral affection, and does not tend to spread to other parts of the lung, as from the upper to the lower lobe or *vice versâ*. Help in diagnosis may be afforded by using the tuberculin test (see p. 228).

Prognosis.—In cases which commence with an attack of acute pneumonia life may be considerably prolonged. The duration of recorded cases has varied from a few months to many years; and provided bronchiectasis do not supervene, the condition is compatible with a fairly good condition of health. In the subacute indurative form the prognosis is bad, health steadily fails, and the illness usually terminates fatally in a few months.

In pneumoconiosis the disease is of slow progress, and, although as time goes on the condition tends to become worse, life may extend over a number of years.

Treatment should be carried out on the lines recommended for chronic pulmonary tuberculosis. The patient should be removed from all unhealthy surroundings, and fresh air and good food are the first essentials. If bronchitis be present, it should have appropriate treatment. Tonics and alteratives, such as iron, quinine, arsenic, and cod-liver oil, are useful. Workmen who are exposed to the inhalation of irritating dust should wear a cotton-wool respirator.

PULMONARY TUBERCULOSIS

The anatomical changes characterising the various forms of pulmonary tuberculosis, together with the associated morbid conditions, have been described at pp. 90-97. The various etiological factors, and especially the part played by the tubercle bacillus, have been discussed at pp. 50-59 in relation to the pulmonary varieties of tuberculosis, and more generally in the article on tuberculosis (Vol. I. pp. 220-234), where a description is also given of the symptoms and course of the acute general form of the disease. Clinically as well as anatomically we may recognise three distinct types of pulmonary tuberculosis. These are (1) an acute miliary form; (2) an acute caseating form; and (3) a chronic form. Either of the acute varieties may arise in a case of the chronic type as well as independently.

ACUTE MILIARY PULMONARY TUBERCULOSIS

The affection of the lung is only part of a general tuberculous infection, and in some cases its symptoms may be overshadowed by those arising from tubercle in the meninges or peritoneum. We shall limit our description to that variety in which the pulmonary affection predominates.

Symptoms. — The invasion of the disease is sometimes insidious, slight cough, loss of flesh and strength, and perhaps sweating at night being the only symptoms present. Such a mode of onset is common in children, who in the early stage are observed to be fretful, languid, and feverish at night, without evident cause. In other cases the onset is more sudden, with rigors, pyrexia, and

prostration, which may or may not be accompanied by thoracic symptoms, such as dyspnoea, cough, hæmoptysis, and pains in the side. In some cases the symptoms are those of pleurisy with effusion, but the fluid tends to disappear early from the affected side to appear on the other. Dyspnoea is the earliest thoracic symptom in a large proportion of cases, and is more commonly objective than subjective. The breathing is often irregular, and attacks of rapid breathing may alternate with periods of normal respiration. The rate of respiration is not infrequently extremely quick, especially in the case of children. Sometimes subjective dyspnoea is marked and the patient may be quite unable to lie down. A high degree of cyanosis is sometimes present, the lips, cheeks, and hands being bluish, and the extremities cold. Cough is an almost constant symptom, but exceptionally it is little marked or absent. There is usually some expectoration, but as a rule it is small in amount. The sputa commonly consist of mucus, and occasionally a little blood is present. Tubercle bacilli are often absent.

The pyrexia in acute miliary tuberculosis is usually of characteristic type, and the temperature is always above normal. Two types are observed, in the first of which the evening temperature is distinctly but only slightly higher than the morning. In other cases the pyrexia is of an "inverse type," the morning temperature being higher than the evening, the remissions being sometimes more marked. Sometimes the inverse type alternates with the first. The pulse is usually rapid, but may become slow on the supervention of cerebral symptoms. Epistaxis and petechial and purpuric hæmorrhages in the skin sometimes occur.

Sweating is common and may be profuse. Sudamina, herpes, and a variety of skin eruptions have been observed. The face is pale or flushed. Emaciation is usually present, but is less marked in the pulmonary than in the cerebral and abdominal forms. As the illness advances, prostration becomes more and more marked, and before the end the "typhoid" condition usually supervenes.

The **physical signs** are not characteristic. The lungs are usually hyper-resonant as the result of associated emphysema. The breath sounds in certain situations may be feeble or interrupted, but as a rule they are obscured by sibilant and sonorous rhonchi. Râles may appear late in the disease. There is sometimes palpable enlargement of the spleen. Ophthalmoscopic examination occasionally reveals the presence of choroidal tubercle.

Prognosis.—In most cases the illness ends in death. Some-

times remissions occur, but these are usually followed by recrudescence of the disease or by evidence of an outbreak in some other part of the body. In rare cases chronic pulmonary tuberculosis supervenes or complete and permanent recovery takes place. The duration of the fatal cases varies from a week to three months and averages about five or six weeks. The prognosis is always extremely grave.

The **diagnosis** is often a difficult matter. The physical signs, as has been pointed out, are not characteristic; and even if expectoration can be obtained, which is seldom possible in the case of children, tubercle bacilli are more likely to be absent than present. In most cases a correct diagnosis cannot be made at an early stage. The most important points are the character of the pyrexia and the degree of dyspnoea and cyanosis. Sometimes the discovery of choroidal tubercles enables one to make a positive diagnosis.

The **treatment** of the acute miliary form of pulmonary tuberculosis must be symptomatic. Abundant fresh air and careful feeding are essential. If constipation be present, aperients should be given. In cases with restlessness and pain, small doses of opium or bromide of potassium may be tried. If the temperature be high, tepid sponging may be employed. If there be marked cyanosis and dyspnoea, oxygen should be administered. Complications must be treated as they arise.

ACUTE PNEUMONIC PHTHISIS

SYN.: ACUTE CASEOUS PULMONARY TUBERCULOSIS, PHTHISIS
FLORIDA, GALLOPING CONSUMPTION

Symptoms.—In some cases the illness commences more or less suddenly, with rigors, fever, and prostration, or with hæmoptysis. Cases which begin acutely occasionally bear a close resemblance to acute pneumonia, the symptoms being dyspnoea, cough, pain in the side, altered pulse-respiration ratio, rusty sputa and the physical signs of consolidation soon appearing. Hæmoptysis may be actually the first symptom, or it may be preceded by rigors or vomiting. Sometimes the illness simulates typhoid fever, prostration, headache, and diarrhoea being combined with pyrexia. More commonly, however, the onset is gradual, with languor, loss of

appetite, gastro-intestinal disturbance, chilliness, aching in the limbs, and cough. Even in cases which appear to begin suddenly it will be found that the patient has been for some time previously in indifferent health. Whether it begin suddenly or insidiously, the illness soon presents the same features. There is pyrexia, sometimes nearly continuous, sometimes remittent, sometimes of an irregular type, but usually characterised by high evening temperatures and morning remissions of two or three degrees. The pulse is generally rapid and the respirations are quickened, but the pulse-respiration ratio is not so markedly altered as in pneumonia. Dyspnoea is present and continuous, though seldom extreme or distressing, and there is a moderate degree of cyanosis. Cough may be little troublesome, or frequent and hacking. The amount of expectoration varies, the sputa being at first probably mucous, later mucopurulent or purulent and of a greenish colour. In rare cases expectoration is altogether absent. Sudden hæmoptysis, as already mentioned, may be the first symptom. Hæmoptysis may be absent throughout, or it may be considerable and frequent, or the sputa may be simply rusty or blood-stained. Tubercle bacilli are nearly always present in the sputa, and elastic fibres may be found in the later stage.

The appetite is usually lost, the tongue becomes furred, and the bowels are either constipated or loose. There is progressive emaciation and increasing weakness, and profuse sweating is common. The patient is often restless and sleepless, and may be delirious at night.

Physical signs.—At first the signs may be simply those of bronchial catarrh, rhonchi being audible more or less generally over the lungs. Signs of consolidation, however, soon make their appearance, usually in the upper, but sometimes in the lower lobes on one or both sides. The area of consolidation extends, and signs of excavation may appear in the midst of a consolidated area. Abnormal physical signs do not disappear from the situations in which they first show themselves, although new parts become affected. There is sometimes albuminuria, and the spleen may be enlarged.

Prognosis.—The prognosis is always grave. The duration of the illness varies, but averages two or three months. In some cases after a time the acute symptoms subside, the fever ceases, and a process of repair progresses in the lungs. The disease may then either become chronic or undergo arrest. In most cases, however, there is no interruption in the downward course. The patient becomes weaker and more prostrate, and gradually sinks, or is

carried off by some complication such as hæmoptysis, pneumothorax, or general miliary tuberculosis.

The most unfavourable cases are those which occur in young adults or adolescents.

Diagnosis.—In cases with sudden onset, acute pneumonia may be suspected, and in cases of apical pneumonia the reverse mistake may be made.

In acute pneumonic phthisis, however, consolidation, as a rule, does not set in nearly so rapidly as it does in pneumonia, and the duration, the course, and the march of the physical signs will usually enable a correct diagnosis to be made. The persistence of pyrexia and the slow extension of consolidation are important points. It must be remembered that in some cases it is impossible at first to distinguish the illness from acute pneumonia.

Cases of empyema after pneumonia are perhaps not infrequently mistaken for acute pulmonary phthisis. The physical signs are, however, those of fluid, not those of consolidation, and in doubtful cases an exploratory puncture should not be omitted.

Treatment.—As the treatment of acute pneumonic phthisis in no way differs from that of the acute stage of chronic phthisis, it will be more convenient to defer its consideration to the next section when dealing with the treatment of the chronic form of the disease.

CHRONIC PULMONARY TUBERCULOSIS

CHRONIC PULMONARY PHTHISIS

Symptoms.—Chronic pulmonary tuberculosis usually begins in an insidious manner, and the early symptoms are often misleading. The illness may commence with failure of general health, weakness, and lassitude, without obvious cause. In other cases the digestive system is the first to be affected, there being loss of appetite, dyspepsia, pain after food, and vomiting, or chronic looseness of the bowels. Anæmia, which does not yield to the ordinary remedies, or amenorrhœa, may afford the first evidence of ill-health. In all these cases, however, if the temperature be carefully watched a certain degree of pyrexia will probably be found. Pulmonary symptoms may be altogether in abeyance, but in many cases cough with some expectoration is present, and there may be little to distinguish the illness from bronchial catarrh.

But the disease not uncommonly commences somewhat suddenly

with hæmoptysis, which may be profuse or only moderate in amount, and may occur without any premonitory symptoms or after a period of ill-defined ill-health, such as has been referred to. Other cases begin with acute pleurisy, or with the signs and symptoms of acute pneumonic phthisis. The chief symptoms of the disease itself are cough, shortness of breath, emaciation, failure of strength, loss of appetite, dyspepsia, fever, night-sweats, anæmia, and quickened pulse and respiration. Some of these are obviously the result of the local lesion, others are referable to toxæmia following on general infection.

Cough is one of the earliest and most constant symptoms, seldom leaving the patient unless the disease become arrested or quiescent, although in exceptional cases it may be very slight or entirely absent. At first it is usually hard, short, and hacking, with scanty mucous expectoration. Later, it may be easy and accompanied with free expectoration, or it may be difficult, coming on in paroxysms which are exhausting and distressing. In some cases it chiefly occurs in the morning, on waking from sleep or while the patient is dressing. It is very often particularly harassing at night, when the patient is composing himself to sleep. In some cases a fit of coughing is excited by food and terminates in vomiting, and thus seriously interferes with nutrition. Cough, as is to be expected, is most troublesome when the disease is active and progressing.

Sputum.—The sputa may be mucous, muco-purulent, or purulent. There is nothing characteristic about the mucous expectoration of the early stages, and it is not until the stage of softening and excavation that the sputa can be said to be typical. They are then thick, greenish-yellow, and nummular or discrete. The sputa are free from fœtor except when bronchiectasis or gangrene is present. Expectoration may be absent or may amount to little more than a drachm in the twenty-four hours, while in advanced cases there may be as much as 10 or 12 ounces. A somewhat rare event is the expectoration of calcareous masses, a sign of a long-standing and formerly arrested lesion. When the disease is active and pulmonary tissue is breaking down, elastic fibres as described at p. 106 may be detected. The most important feature of the sputa is the presence of tubercle bacilli, which are only found in cases of pulmonary tuberculosis. The method of mounting and staining the sputum for tubercle bacilli is described at p. 108. It must not be concluded that there is no pulmonary tuberculosis if bacilli be absent, but if on repeated examination no tubercle bacilli be found it is unlikely that the case is one of active pulmonary tuberculosis.

Hæmoptysis occurs at some stage of the disease in at least 50 per cent of the cases. Its direct and indirect causes, its characters, its significance, and the conditions likely to be mistaken for it have been considered at pp. 108-110. It is more common in the later than in the early stage, in males than in females, in middle life than in youth and old age, and in hot weather than in cold. In certain cases hæmoptysis occurs again and again during the course of the illness. The amount of blood brought up may amount to only a few streaks or be just sufficient to colour the sputa. In other cases small quantities varying from a fluid drachm to an ounce may be repeatedly expectorated, or large amounts from ten to thirty ounces may be brought up at one time. Hæmorrhage may be the first symptom, occurring without obvious cause in an apparently healthy person; but although it may be large in the early stages, it very rarely then proves fatal. It is more serious as well as more frequent in the later stages, and a large hæmorrhage is then not very uncommonly the direct cause of death. In some cases pain in the side or shoulder or at the apex of the lung precedes hæmoptysis, on the occurrence of which it subsides.

Shortness of breath is a very constant symptom. It is readily induced by exertion or excitement, while it may not be noticeable when the patient is at rest, although even then the breathing is probably quicker than normal. A marked degree of cyanosis is unusual except in cases where the disease is basic or affects a large tract of lung, or when some complication such as pneumothorax is present. Clubbing of the fingers may often be observed in chronic cases, but it seldom attains such a degree as it does in some other diseases, such as bronchiectasis.

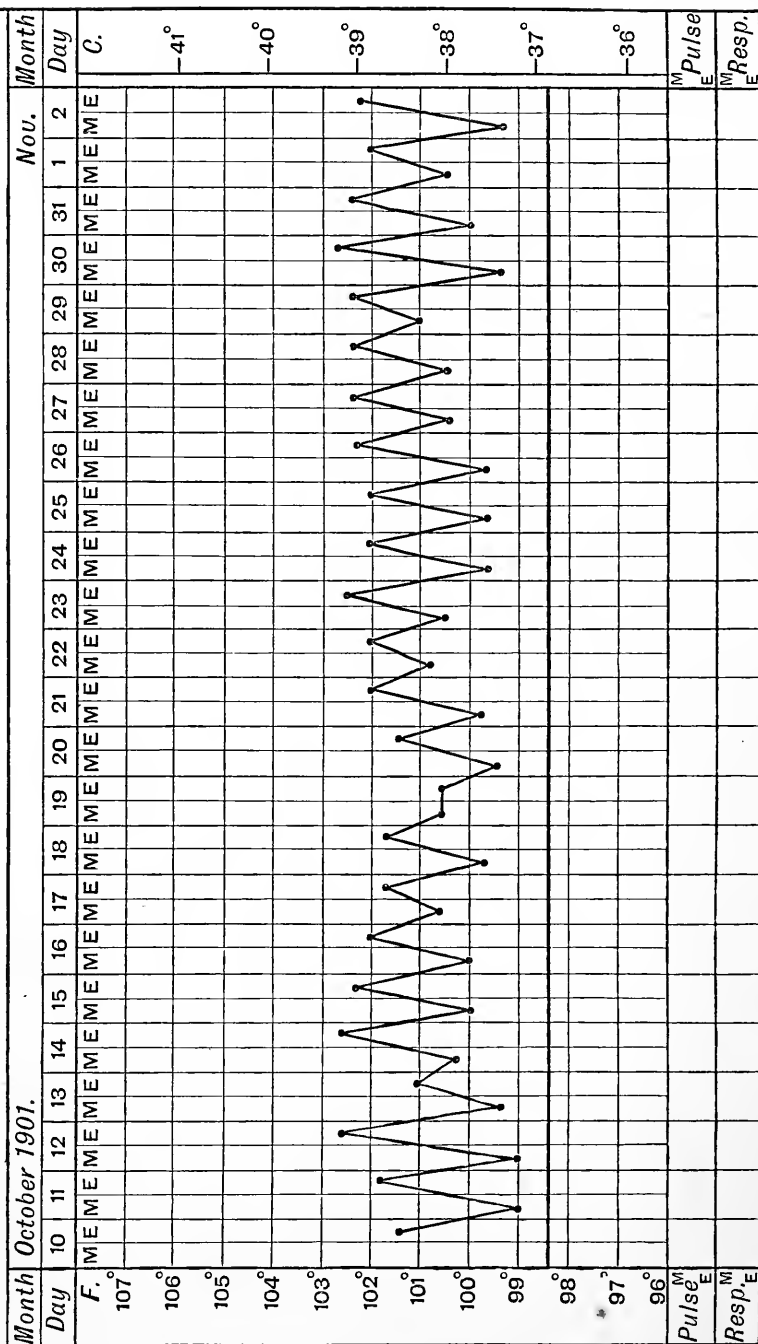
Emaciation.—When the disease is active there is usually wasting, which in advanced cases may be extreme. Much can be done by judicious treatment to improve the state of nutrition. Under favourable conditions weight may be steadily gained, and with arrest of the disease may reach and be maintained at a much higher level than previous to the illness. It should be observed that although gain in weight is usually a good sign, it is quite possible for the patient to put on flesh while the disease in the lung is advancing and no improvement is taking place in other directions.

Loss of energy and debility are marked symptoms of the active stage of the disease. In advanced cases there is usually great weakness and prostration.

Pain in the chest is not infrequent, and may be due to pleurisy or to intercostal neuralgia, or to the straining caused by coughing.

Temperature Chart in a case of Caseating Pulmonary Tuberculosis.

Female aet 41.



Local pain or a sense of soreness may be felt in the affected apex. According to Dr. Head, referred unilateral pain over the front and back of the chest, with more or less superficial tenderness over the same side of the chest and scalp, occurs in cases of a bronchitic type. Cases of rapid, extensive consolidation or excavation are usually without referred pain. Aching in the back and limbs is common in febrile cases. The muscles are sometimes tender when touched, and percussion may give rise to small, transient, local swellings, to which the term "myoidema" has been applied. These are not peculiar to phthisis, but may be observed in most wasting diseases.

Digestion.—The tongue is often slightly furred or may be thickly coated. Loss of appetite and dyspepsia may be the first symptoms of illness, and are common at all stages. In some cases vomiting is troublesome, especially after meals, following a fit of coughing, or occurring independently. The bowels are often irregular, constipation being the rule at an early period, while looseness is common in the later stages.

Fever.—Fever is nearly always present in some degree at the onset of the disease, and is a frequent incident in all its stages. Pyrexia is usually marked, slight, or absent, according as the disease is active, stationary, or arrested. There are two important types of fever observed when the disease is active. In one form, which is characteristic of miliary tuberculosis, fever is more or less continuous, with but little difference, not more than one or two degrees, between the morning and evening temperatures (see Chart, p. 216). In the other, which is typical of caseation, there are marked remissions of two, three, or more degrees (see Chart, p. 222). In either case the evening temperatures are usually the higher; but exceptionally, particularly in cases of miliary infiltration, an inverse type is met with, when the morning temperatures are the more elevated. It is about 8 A.M. that the temperature is lowest and about 8 P.M. that it is highest. In chronic cases with little activity the temperature curve may over a considerable period of time show slightly pyrexial evening temperatures, 99.8° or 100.2° F., while the morning temperatures are subnormal, 97° or 98° F. Professor Koch is of opinion that pyrexia ranging over 100.4° F. results from the invasion of septic micro-organisms, but at present this is a mere theory. In chronic cases which have undergone arrest the temperature is sometimes over long periods subnormal. A low temperature with general signs of improvement is favourable, but a subnormal temperature with signs of progress of the disease is of bad omen. In chronic cases periods

of pyrexia often alternate with periods of apyrexia, and we can learn something of the nature of the changes taking place in the lungs by a study of the temperature chart, which in some cases points to miliary infiltration, in others to caseating pneumonia.

Exercise and excitement are apt to raise the temperature two or three degrees except when the disease is quiescent, and in individual cases the effect of exercise on the temperature should always be carefully watched.

Sweating at night is common when the disease is active and some degree of fever is present. It usually occurs in the early morning, at the time when the temperature is falling, the patient waking out of sleep to find himself bathed in perspiration. Sweating is, however, to a large extent dependent on the conditions under which the patient lives, and is promoted by impure air at a temperature over 60° F. and an excessive amount of bedclothes. Such sweatings, often erroneously regarded as a cause of weakness, are really the result of a lowered tone of body. Profuse perspirations are usually accompanied with marked prostration, and are characteristic of advanced disease. Pityriasis versicolor is often observed among hospital patients.

The pulse is generally accelerated, except when the disease is quiescent. It is rapid, not only when there is fever, but often when there is no rise of temperature. It is almost always over 100 when the disease is advancing. The heart even in apparently quiescent cases is unduly irritable, and slight exertion or excitement suffices to bring on rapid action. Sometimes there are attacks of palpitation, especially after food. Cardiac irritability is more marked in males than in females. A habitually normal pulse rate is a very favourable sign.

Anæmia is usually a more or less marked symptom, and in a typical case the patient is pale and has a hectic flush. The red blood-corpuscles are not uncommonly diminished in number and deficient in hæmoglobin. Apart from this the blood changes are unimportant. The blood platelets are more abundant than usual, and leucocytosis may be observed after hæmoptysis and in cases of caseous pneumonia. A slight degree of œdema of the legs is not uncommon.

There are no special *urinary symptoms*. In the later stages there may be albuminuria, which is frequently due to amyloid disease of the kidneys.

In females the *catamenia* are usually irregular, and menstruation is often in abeyance while the disease is active. Pregnancy generally

runs its full course, often with some temporary improvement in the disease, which, however, makes rapid progress after delivery.

The *nervous system* as a rule is unaffected. Neuralgic pains sometimes occur in various parts of the body, and neuritis has occasionally been observed, but is decidedly rare apart from alcoholism or other direct cause. Complications affecting the nervous system, such as meningitis, sometimes terminate the illness. It is characteristic that the patient is usually hopeful to the end, a condition to which the name "*spes phthisica*" has been given. The mental faculties remain unimpaired, and great intellectual ability sometimes goes along with pulmonary tuberculosis. Certain mental changes occasionally are observed, usually in cases where there is reflected pain of some intensity and duration, states of depression, exaltation, or causeless suspicion among others (see p. 120).

Physical signs.—It is only by means of physical examination that we can gain any really trustworthy knowledge as to the extent and nature of the pulmonary lesions. While the apices of the lungs are usually the earliest parts to be affected with tubercle, it should be borne in mind that sometimes the signs of disease are most marked posteriorly in the supra-spinous fossæ, and in all cases these regions, as well as the interscapular spaces, should be as carefully examined as the front. Examination should always be made in the standing or sitting as well as in the recumbent position. In cases of bronchitis, pneumonia, pleurisy with effusion, or recent hæmoptysis, a guarded opinion should be given as to the state of the lungs with respect to tubercle. The characteristic physical signs are those of consolidation, of softening, or of excavation. Miliary infiltration does not generally give rise to typical signs, and it is important to remember that early in the disease physical examination may reveal nothing abnormal.

In early cases there may be a slight impairment of resonance or definite dulness at one or other apex, in front or behind, or in the second intercostal space near the sternum. The breath sounds in the same situations have in some cases a wavy, interrupted character, or may be feeble or bronchial, and the respiratory movements are deficient. Much weight should not be attached to a slight degree of dulness at the right apex in either sex or to harsh breathing with prolonged expiration in the same situation in a woman. Sometimes there are signs of bronchial catarrh, but the rhonchi are after a time limited to one apex. The most important sign, however, in the early stage is the presence of crepitations or

râles, which at first may be audible only on deep inspiration or after cough. The râles are most commonly of a dry, crackling character. When arrest occurs at this stage, the breath sounds are commonly weak at the affected apex, and the percussion note may be either hyper-resonant from emphysema or normal or slightly dull.

With marked consolidation there are the usual signs, diminished movement, dulness, increased vocal fremitus, bronchial or tubular breathing, bronchophony, and pectoriloquy; and when there is secretion in the tubes, crepitations are audible, which have a peculiar bright or clear quality, the so-called "consonating" râles. Abundance and coarseness of râles point to softening, which is also indicated by the presence of elastic tissue in the sputum.

Cavities do not always give rise to characteristic physical signs. They must be of a certain size, not smaller than a walnut, at least partially empty, and not very deeply seated. In the case of a cavity of fair size and near the surface there is usually some retraction of the chest-wall, but in rare cases slight bulging has been observed. The movements of the chest-wall are diminished, but not to so great an extent as over-consolidated or fibrotic lung. Recession of the chest-wall has been noticed at the commencement of inspiration, and bulging sometimes occurs during the act of coughing. The sound produced by percussion over a cavity varies in different cases. Sometimes it is so typical of a cavity that it may be called cavernous. The percussion note has then a peculiar hollow quality and a pitch which varies with the dimensions of the cavity, being higher the smaller its size. It was pointed out by Wintrich that the pitch of the cavernous percussion note in certain cases is raised when the mouth is opened. Friedreich observed that the pitch became higher when percussion was made during deep inspiration; and Gerhardt noticed that it was altered with change in the patient's position, as the result of the displacement of the fluid contents of the cavity. In addition to being hollow the note has sometimes a peculiar cracked quality, the cracked-pot or *pot fêlé* sound (see p. 129). Both the hollow quality and the cracked-pot sound are often better brought out when the patient opens the mouth while the chest is percussed. If the cavity be full of secretion or be separated from the surface by consolidated lung or much-thickened pleura, the note will be simply dull. When the cavity is very large and superficial, the bell sound or *bruit d'airain* may be obtained. The vocal fremitus over a cavity is increased. The breath sounds may be tubular, but in typical cases they are

cavernous or amphoric (see p. 132), a hollow quality being added to the tubular character, and the expiration being of a lower pitch than inspiration. Unless the cavity be dry, coarse bubbling râles of a metallic or cavernous quality are usually audible, and sometimes there is distinct metallic tinkling. The sounds of the voice and cough are conducted to the ear with increased distinctness, and pectoriloquy is usually present. In certain cases after cough there is a peculiar sucking or hissing sound, due to sudden inrush of air into the cavity. This sound, which has been called the "post-tussive suction sound," is decidedly uncommon. When a cavity is situated in the neighbourhood of the heart, the impulse of the heart on the cavity sometimes produces a systolic whiffing murmur or gives rise to clicks or sounds of a metallic character synchronous with the beat (see p. 137).

In cases of fibrosis there is often considerable diminution in bulk of the pulmonary tissue, as the result of which the affected side is retracted or actually depressed, while the inspiratory expansion may be altogether abolished. In some cases the note is dull and wooden in character and there is marked percussion resistance. In other cases, however, the fibrosis is combined with emphysema, so that there is but little alteration in resonance. The breath sounds may be weak or entirely suppressed, but bronchial breathing is present if the tubes remain patent. The vocal fremitus is usually increased. Fine crackling râles are often persistently audible over a considerable area, and creaking sounds due to pleural adhesions are of common occurrence. Fibrosis is frequently combined with excavation, and in such cases characteristic changes in the chest-wall may be noticeable on inspection. There is flattening or sinking-in of the chest-wall, together with impairment or loss of movement in the situation corresponding to the lesion. When the disease is in the left lung, the heart may be uncovered and drawn over to the left, its pulsations being visible in the second and third interspaces, while the impulse may be felt in the anterior axillary line. When the disease is in the right lung, the pulsation of the aorta may be seen and felt in the second right interspace, which in some cases has given rise to the suspicion of aneurysm.

Complications.—The principal complications are either caused by the local process in the lungs or result from the invasion of other parts of the body by tubercle. The anatomical changes and pathology of these complications have been considered at pp. 93-97. The local disease may lead to the rupture of a blood-

vessel and hæmoptysis, which has already been considered, or to perforation of the pleura and pneumothorax, which is discussed at p. 268. The most important secondary tuberculous lesions are laryngeal tuberculosis, pleurisy, pericarditis, ulceration of the intestines, peritonitis, meningitis, and general miliary tuberculosis. These add their own dangers to that of the original disease. Anal fistula is an occasional complication. Lardaceous disease and albuminuria occur in advanced cases, and thrombosis of the veins, especially those of the lower extremities, is not uncommon as a terminal process.

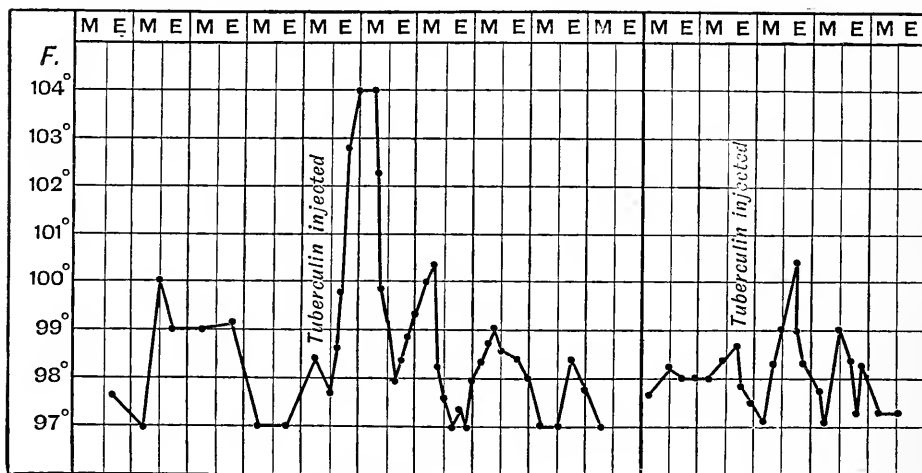
The **diagnosis** of chronic pulmonary tuberculosis presents its chief difficulties in the early stages when physical signs are but little marked, but it is then that it is of the greatest importance to attain certainty as to the nature of the case, and that the disease is specially amenable to treatment. The symptoms of the early stage, as already mentioned, may mislead. In cases where there are cough and expectoration the examination of the sputum for tubercle bacilli may at once establish the diagnosis. While the presence of tubercle bacilli is a certain sign of pulmonary tuberculosis, their absence does not negative this disease, as already stated, unless repeated examinations have been made. In many cases, however, no expectoration can be obtained. A careful record of the temperature is in such cases of great diagnostic importance; and if it be found that the evening temperatures are above normal, while the morning temperatures are a degree or two less, the probability of tuberculosis is great. In doubtful cases, where the temperature is normal and the diagnosis cannot be settled by sputum examination, the tuberculin test may be tried. The physical signs in early cases may be very indefinite, and it may be only by carefully comparing one side with another, and by making several examinations, that such signs as have been described can be detected. When definite physical signs of consolidation, softening, or excavation are present, over one or both upper lobes, the stage of difficult diagnosis is usually past. The difficulties with regard to chronic bronchitis and bronchiectasis have been already alluded to, and those connected with syphilis, hydatid, and new growth will be subsequently considered.

Tuberculin as a diagnostic agent.—For diagnostic purposes, Koch's original tuberculin should be used. Tuberculin is a brownish liquid, an extract of pure cultures of tubercle bacilli, containing about 50 per cent of glycerin. Pure cultures are grown in veal-broth rendered slightly alkaline, to which 4 to 5 per cent of

glycerin has been added. The culture medium is placed in shallow vessels, and the growth spreads over the surface, forming a whitish-gray layer. The medium with its culture is then evaporated to one-tenth of its bulk over a water-bath at boiling-point, and is subsequently filtered through porcelain. Any bacilli it may contain are killed by the action of heat. In making use of the test, it must first be ascertained that the patient's temperature is normal. The patient should be kept in bed for forty-eight hours, the temperature being taken every four hours. If the temperature during that time prove to be normal, and the patient be not weakly, one milligram of tuberculin is injected. If there be a rise of temperature of one degree or over within the following twenty-four hours, the reaction is positive. If there be a slight rise of only half a degree, the same dose should be repeated after an interval of two days, when a more marked reaction may take place. Should there be no rise of temperature after the first, or only a slight rise after the second injection, two milligrams should then be injected. If a doubtful reaction occur, the dose should be repeated after a short interval, as before. If still no certain reaction take place, five milligrams should be injected. If after that doubt should remain, ten milligrams may be injected twice. In the case of a child under five, or a weakly subject, the initial dose should be a quarter of one milligram; and in the case of a child between five and ten, half of one milligram. Any marked rise of temperature is accompanied by some constitutional disturbance, shown by a feeling of malaise and pains in the back and limbs. Sometimes vomiting occurs, and sometimes there is a general erythema. Besides a general reaction there is a local one in the lung, shown by increase in cough and expectoration, and sometimes temporary increase in râles, just as in the cases of superficial tuberculous lesions, local swelling, redness, and pain are produced.

The injections should preferably be made into the back or buttock, and the time chosen should be either late in the evening or early in the morning, as the rise of temperature usually does not occur until some ten hours after the test is applied. The required dilution is made by adding 1 part of tuberculin to 100 parts of sterilised water, containing 10 per cent of glycerin and .5 per cent phenol. One milligram of tuberculin will be contained approximately in $1\frac{1}{2}$ minims of this dilution. The charts which are appended are examples of well-marked reactions. Although the value of a positive reaction from a diagnostic point of view is very great, the test is not infallible, as exceptionally reactions occur in

non-tuberculous subjects. When the test is thoroughly made and no reaction occurs, it is extremely improbable that the case is one of tubercle.



Temperature charts in two cases showing the tuberculin reaction

Serum diagnosis.—Messrs. Arloing and Courmont have endeavoured to render practical a method of serum diagnosis in tuberculosis. They first obtained homogeneous cultures by special methods, the media being shaken every day during the period of growth. By adding to the cultures 1 per cent or half of 1 per cent of formalin, they found they were able to preserve them, while the power of agglutination was not affected. At present this method of diagnosis has not been sufficiently tested to enable us to form an opinion as to its value. It is maintained by Messrs. Arloing and Courmont that in tuberculous subjects a dilution of from 1 in 5 to 1 in 20 will produce agglutination, except in cases of severe infection or extensive lesions.

The Röntgen rays, when employed by a skilled observer, are useful helps to diagnosis. Although at present the first beginnings of tubercle in the lung cannot be discovered by their means, tuberculous consolidation can sometimes be recognised at an earlier period, and a smaller cavity can be detected, than by means of auscultation and percussion.

Course and prognosis.—The course of chronic pulmonary tuberculosis varies considerably in different cases. In some cases the illness may make comparatively rapid progress, terminating fatally in a few months, and in others it may last for many years. The disease sometimes remains almost altogether latent. Tubercles

may form in the lungs, give rise to few or no symptoms, and undergo arrest, as is shown by the presence of healed lesions in the lungs of persons dying from some other cause who have not been known to have had any pulmonary trouble (see p. 96). In some cases the disease is steadily progressive until it terminates in death. In others there are successive outbreaks of activity, during which the disease in the lung extends, followed by intermissions of longer or shorter duration, when the morbid process appears to be stationary. In some cases the disease after a certain time undergoes arrest, and the patient may remain in good health for a number of years.

There is no disease in which it is more difficult to forecast what is going to happen, and mistakes must necessarily be made, even by those of great experience. Many factors have to be considered in forming a prognosis. It goes without saying that generally the earlier the stage of the disease and the less the extent of lung affected the more hopeful is the outlook, and that the opposite holds true. Signs of actively progressive disease with profuse expectoration are bad, while evidences of fibrosis or of a contracted and dry cavity are good. Slow progress of the disease is a good sign, and, if after a duration of some years the disease has not produced much damage, it is probable that its future course will be favourable. A good appetite, a sound digestion, regularity of the bowels, absence of pyrexia, and a quiet pulse are all good signs. A stationary weight with a good state of nutrition, or an increase when the weight is below normal, is good. If the general constitution and physique be good, and the habits satisfactory, the case is more hopeful than when the patient is a weakling, or has led a life of dissipation. When the disease shows itself soon after puberty, it is often of a severe type. After the age of forty-five the progress of the disease is generally slow. Women on the whole do not respond to treatment so well as men, and in them the dangers of the disease are greatly increased by child-bearing. When the disease has run a favourable course in other members of the family, it is a hopeful sign, and the reverse holds true. Continued pyrexia, especially of the types described as associated with miliary infiltration and caseation, is unfavourable. Persistent diarrhoea is a bad sign, and peritonitis usually terminates in death. The complication with laryngeal disease is always grave.

The financial position of the patient often materially affects the prognosis, for the possession of means will enable him to command all the advantages of climatic and hygienic treatment for a prolonged period. In every case there is some degree of uncertainty,

and such complications as acute miliary tuberculosis, hæmoptysis, or pneumothorax may unexpectedly imperil life at any stage in a case otherwise favourable. Hæmoptysis is most serious in the later stages. Cases in which hæmoptysis occurs early in the disease, except those of a pneumonic type, usually run a favourable course. Pneumothorax is fatal in three cases out of four, and when it affects the less diseased side the patient very rarely recovers.

Prophylaxis.—The chief way in which tuberculosis is disseminated is by means of the sputum (see pp. 57, 58), and if the expectoration can be prevented from becoming dry, and be destroyed by burning, or removed by pouring it down a drain, no infection can take place. The following are the rules drawn up by the National Association for the Prevention of Consumption:—"The consumptive person must not expectorate about the house, nor on the floor of any cab, omnibus, tram-car, railway carriage, or other conveyance. Spitting about the streets, or in any public buildings (churches, schools, theatres, railway stations, etc.), is as dangerous as well as a filthy habit. The consumptive person must not expectorate anywhere except into a special vessel or cup kept for the purpose, and containing a little water. When out of doors, a small, wide-mouthed bottle with a well-fitting cork may be used; or a pocket spittoon, which may be obtained from any chemist. For wiping the mouth a rag or paper, which can afterwards be burned, should be used instead of a handkerchief. The collected expectoration must be carefully burned on the back of the fire, at least once daily; this is the simplest, quickest, and safest way of destroying the germ. When there is no fire, the expectoration must be washed into the drain or buried in the earth. The cup or spittoon must then be well washed with boiling water. When not provided with a proper vessel, a consumptive person must not spit into a handkerchief, but into a piece of rag or paper, which must be burned. Consumptive persons must not swallow their expectoration, as, by so doing, the disease may be conveyed to parts of the body not already affected. A consumptive person must not kiss, or be kissed, on the mouth."

No one who has any sign of active tuberculous disease should marry, and no one in whom the disease has been arrested should marry unless it be possible to live an open-air life. It is advisable that a tuberculous person should have a separate bed, and, if possible, a separate room. When a consumptive patient becomes confined to bed, great care should be taken to prevent the bedding and floor being soiled with expectoration. The room should be

kept thoroughly clean, and should be cleansed with wet dusters. Every room in which a consumptive person has lived or died should be disinfected and thoroughly cleaned before it is again inhabited. The dangers of milk and meat can be prevented by thoroughly boiling the milk and thoroughly cooking the meat.

Treatment.—We shall consider the subject of treatment under the following headings: hygienic, climatic, specific, medicinal, and symptomatic.

Hygienic.—In all forms and stages of the disease it is of the first importance to place the patient under the best hygienic conditions; and the most effective of all remedies are fresh air, sunlight, good food, and rest, combined with exercise when the patient is able to take it. It is only within comparatively recent years that the vital importance of fresh air for the consumptive invalid has been generally recognised. The conditions under which the patient was formerly placed were highly artificial, if not actually unwholesome. It was considered that the patient must, above all things, be protected from chill and draughts, there being the greatest dread lest he should catch cold. It was held that it was more important to attend to the temperature of the air than to secure its purity. The patient, when unable to go out of doors, was accordingly confined to a room with windows kept shut by day as well as by night, the temperature being maintained at 62° F. or higher. Although it was well known that phthisis was most prevalent in ill-ventilated and over-crowded dwellings, little was done to see that the sick person was placed under much better conditions. This plan, as was pointed out long ago by Dr. Graves of Dublin, only tended “to make the constitution delicate and consequently more liable to the inroads of phthisis.” It has now been conclusively proved that not only is the tuberculous invalid extremely tolerant of fresh air, but that he never progresses so well as when he can be supplied with fresh air to breathe by night as well as by day. Whether the patient is confined to bed or is able to be up, he should, during the whole twenty-four hours, have air to breathe as nearly as possible as pure and as fresh as the outside air. While dust and smoke and smoke-laden fog are injurious and should be avoided, they are much less harmful than air which is polluted with organic impurities. As the air of towns can never be as pure as the air of the country, it is desirable that the consumptive patient should live in the country; but whether he be in town or in the country, the windows of the rooms he occupies should be kept widely open night and day.

It is still better when it can be arranged that the patient should live altogether out of doors, sleeping on a verandah or balcony, or in a shelter properly protected from wind, rain, and cold. Sunlight and sunshine have important influences on the blood and the tissues, and, when the sun's rays are not too powerful, the patient should be allowed to bask in the sun.

Rest.—The patient should be kept in bed continuously if the temperature be up in the morning as well as in the evening, or if the temperature in the evening rise above 101° F. The presence of pyrexia is one of the main indications for rest. Constant rest is also necessary if the pulse be persistently above 100. In all cases where there is any sign of active disease the patient should make a point of resting in the horizontal position for half an hour before and for half an hour after each meal.

Exercise.—Exercise must be carefully regulated, according to the patient's strength and powers of endurance. As a rule, walking is the best form of exercise as long as the disease is still in progress. The distance and the rate of walking will vary with different cases. At first very short walks on the flat only should be allowed. The temperature should be taken immediately after exercise, and if it have risen more than two degrees the exercise has been excessive. The longer walk, which should be taken in the morning between breakfast and the mid-day meal, will vary from one-half to six miles, and the rate from one and a half to three and a half miles an hour. The afternoon walk should be only half as long as the morning walk. Brehmer and other German authorities attach much importance to hill-climbing in virtue of its effects on the heart. Hill-climbing, when permitted, must be done very slowly and gradually, so that the heart may be strengthened but not strained. All forms of violent exercise must be rigidly prohibited to any one whose disease is not arrested. Cycling, swimming, running, skating, tobogganing, etc., cannot with safety be allowed while there are signs of active disease. Some forms of breathing exercises are usually beneficial in strengthening the respiratory muscles and in securing full expansion of the lungs, but such exercises should always be taken in the open air. For persons in whom the disease is arrested, riding is an excellent form of exercise. Sydenham said of riding that it was the best remedy for consumption, provided it were long enough continued, and he considered the cure depended wholly upon exercise; a doctrine both erroneous and dangerous.

Food.—The dietary should be liberal, and the patient should be encouraged to eat an adequate amount. The open-air life and the

rest before meals help to bring back the appetite. With a little management it will be generally possible to get the patient to take a much larger quantity of food than he has been in the habit of doing. An excessive quantity is neither necessary nor desirable. The diet should consist of plain, wholesome, nutritious, well-cooked, and appetising food; bacon, eggs, milk, butter, bread, fish, beef, mutton, fowl, game, potatoes, green vegetables, and milk puddings being liberally supplied. There is often a marked distaste for fat, but fat is a most important part of the food, and an endeavour should be made to ensure that the patient takes an adequate amount. Koumiss, fermented mare's-milk, and kefir, fermented cow's-milk, as made in steppes of the Kirghis in Southern Russia and in the Caucasus, have been reputed to form a valuable food for the tuberculous. They have a somewhat unpleasant acidulous taste, but form a nourishing and stimulating food. The koumiss to be obtained in this country is fermented cow's-milk artificially sweetened.

Alcohol is seldom necessary in any but the last stages of the disease. If a patient be accustomed to take wine, beer, or spirits in moderate quantities with his meals, there is no reason why he should not continue to do so. All stimulants between meals should be prohibited.

Clothing.—When the patient is confined to bed he must not be overweighted with bedclothes, but must have just sufficient to prevent him from feeling cold. In very cold weather his hands and ears should be protected. Woollen gloves made so as to enclose all four fingers are the most comfortable. When up he should wear woollen underclothing, thick in cold weather, thin in summer. An overcoat should only be worn when standing or resting, and when resting out of doors or by an open window rugs should be used in cold weather.

Baths.—Shower-baths and cold baths followed by a good rub-down with a rough towel are useful tonics for the more robust patients. In all cases it is important to keep the skin in a healthy condition.

Hygienic treatment can be more satisfactorily carried out at a sanatorium than at home, provided the patient be not entirely confined to bed. In the latter case it may be possible to do as much for the patient at home as at a sanatorium. There are now a great many sanatoria for paying patients in various parts of the United Kingdom, and there are many similar institutions in Germany, Switzerland, France, and the United States. The

great advantage which sanatorium treatment presents over ordinary hygienic treatment is that the patient is under constant medical supervision. He is seen three times a day by the resident doctor, and day by day his mode of life is mapped out for him. The patient who is not under such close supervision is constantly doing the wrong thing. He over-exerts himself at one time, and takes too little exercise at another. He engages in exciting games or talks too much. He neglects to keep his windows open, and he perhaps goes to entertainments or lingers in rooms where the air is not fit to breathe. He lacks the constant encouragement to follow a life of physiological righteousness. At the sanatorium he recognises that he is living with one object—that of getting well; and he sees the good effects of the treatment in others as well as in himself. The results obtained by sanatorium treatment are superior to those of any other form of treatment, but it must be clearly understood that it is only in early and limited cases that perfectly satisfactory results can be obtained. The minimum period of treatment at a sanatorium should be six months, but in many cases a much longer period is required. The disease may be considered to be arrested when expectoration has ceased or when bacilli have entirely disappeared from it, when symptoms are altogether absent, and when there are no adventitious sounds on auscultation.

Occupation.—A person who has recovered under treatment up to the point of complete arrest is likely to relapse if he resume an indoor occupation in an unhealthy atmosphere. He should therefore attempt to obtain outdoor employment in order to keep well.

Climatic treatment.—Great importance has in the past been attached to the effect of climate on pulmonary tuberculosis, and there is no doubt that much can be done by the selection of a suitable climate to promote the health of tuberculous invalids. But it has been well said that “care without climate is better than climate without care.” Far more depends on the mode of life than on the climate itself. There are three different kinds of climate to which phthisical patients have been sent to seek health. These are marine climates, high altitudes, and dry, warm climates.

It may be pointed out at the outset that it is our opinion that no one should be sent far from home with advanced disease, or with signs that the disease is in an active condition, as shown by pyrexia and rapid cardiac action. The time to obtain benefit from change of climate is when the disease has become comparatively quiescent. Wherever the patients are sent, they must clearly understand that they are not there to amuse themselves. In many

health-resorts there are unfortunately many temptations to over-exertion and dissipation.

Marine climates are illustrated by many seaside resorts on the south coast, such as Folkestone, St. Leonards, Eastbourne, Brighton, Bournemouth, Torquay, Falmouth, and Penzance. These places are on the whole milder and sunnier during winter than inland resorts. Chronic cases with bronchial catarrh derive most benefit. Winds are sometimes trying, and are especially bad because they drive people indoors. Unless hygienic treatment be thoroughly carried out, no curative action can be expected from residence at the seaside. More advanced chronic cases with bronchial catarrh may benefit at such places as Madeira or Malaga.

Sea-voyages were at one time much in vogue in the treatment of consumption; and although occasionally patients did remarkably well, there is no doubt that many very unsuitable cases were thus sent away from home. Sea-voyages are contra-indicated in all advanced cases, and in all cases in which there are signs of active disease. A voyage through the Red Sea is at certain seasons of the year fraught with considerable danger to phthisical patients. In the case of patients with quiescent disease who are in good general condition, a sea-voyage may sometimes be recommended. The voyage should, however, be in temperate, not tropical, climates; an airy and well-ventilated cabin should be secured; the food should be good, and the patient should be known to be a good sailor. A voyage to Australia by the Cape and back, starting in the autumn and returning in the spring, is one of the best sea-trips. It is important, however, that the patient follow much the same laws as regards rest and exercise as have been already indicated.

The high altitudes, as exemplified by Davos, Arosa, and St. Moritz, have special advantages as winter resorts, inasmuch as the air is usually still, as well as rarefied, dry, and free from dust and micro-organisms. There is, moreover, a comparatively large amount of bright sunshine. Although the temperature is low, the air being still and dry, the cold is well tolerated, and the sun's rays have much warming power. The circulation and respiration are at first quickened and afterwards slowed. The inspirations are deeper and longer, and in course of time the thorax becomes generally increased in size. Metabolism is more active, the appetite is sharpened, the whole body is stimulated, and there is a considerable increase in the number of red blood-corpuscles.

The high altitudes are best adapted for persons under forty who have been of robust constitution, and in whom there are no

acute symptoms. They are contra-indicated in elderly persons, or persons peculiarly sensitive to cold, or of feeble circulation, low digestive powers, or small lung capacity. Cases with emphysema, or of an advanced type with bronchial catarrh, are quite unsuitable. Laryngeal cases, cases with diarrhoea or albuminuria, rheumatic subjects, or those with great nervous irritability, or troubled with insomnia, generally do badly. The fact of a patient having had hæmoptysis is no contra-indication, provided the disease be not far advanced. The patient should go in October, and return to England in April.

The dry, warm climates are illustrated by the Riviera and Egypt. The most suitable places are Hyères, Cimiez, Mentone, and San Remo in the Riviera, Helouan and Mena House in Lower Egypt, and Luxor and Assouan in Upper Egypt.

The Riviera is sunny and comparatively dry, although moister than the desert. Moderate warmth alternates with cool winds. Egypt is warmer during the day, but may be very cold at night, and there are sometimes high winds accompanied with dust. Persons with feeble circulation and advanced in years are likely to benefit. The difficulty with younger patients is that they will seldom take sufficient care, and are apt to commit innumerable acts of imprudence. The percentage of cases of improvement is comparatively small, and cases of deterioration are not uncommon. There is seldom evidence of arrest as the result of residence in Egypt, nor are patients rendered more vigorous.

No one nowadays should be sent to the Cape or to Australia unless the disease is arrested.

Specific treatment.—Tuberculin, which has proved so valuable in diagnosis, was originally introduced as a curative agent, and some, including Professor Koch, continue to believe in its curative action. When used for therapeutic purposes, it is important to avoid as far as possible any local or constitutional reaction. Hypodermic injections of $\frac{1}{100}$ th of a milligram may be given at first, and gradually increased as long as no reaction occurs. Under this plan of treatment great benefit is said to have been observed in early and uncomplicated cases. Professor Koch's new tuberculin has not fulfilled expectations as to its therapeutic value. Serum treatment for pulmonary tuberculosis has been very doubtfully successful.

Medicinal treatment.—Cod-liver oil is a remedy which is of decided therapeutic value in phthisis. It promotes nutrition and improves the condition of the blood, and diminishes the susceptibility to cold. The principal drawbacks to its use are its unpleasant

lishy taste and its tendency to cause eructations. Many persons have an insuperable objection to it. Children, however, as a rule, take it well; and if a carefully prepared oil be used, most adults have little difficulty in taking it. Small doses of one or two teaspoonfuls should be given two or three times a day half an hour after meals. Mixtures with malt extract are palatable, but it should be remembered that they usually contain less than 20 per cent of cod-liver oil. Emulsions of cod-liver oil always contain less than 50 per cent of oil, and their only advantage over oil is their palatability. Pancreatic emulsion, dose 1 to 3 drachms, may be given to persons who cannot take oil. Petroleum emulsion, which is sometimes given, has probably neither nutritive value nor therapeutic action.

Arsenic has long been used in the treatment of pulmonary tuberculosis, and appears to promote nutrition and improve the general health. It is usually given in the form of liquor arsenicalis in doses of 2 to 5 minims after meals three times a day. Cacodylate of sodium, $\text{Na}(\text{CH}_3)_2\text{AsO}$, has recently been recommended by Gautier. It is claimed for it that it is more readily assimilated and less toxic. It may be given by the mouth in doses up to $\frac{3}{4}$ grain, or 5 per cent solution of cacodylate of sodium in sterilised distilled water may be employed hypodermically, doses up to 10 minims being given three times a day. The treatment should be carried on for a week, then suspended for a week, and then resumed.

More recently still, a 5 per cent solution of disodium-methyl arsenate has been employed in the same doses with greater success. Care should be taken in the administration of arsenic to avoid gastric disturbance, and its use should be suspended should toxic symptoms arise.

Creosote and various derivatives from it have for many years been used in the treatment of pulmonary tuberculosis. Creosote has no specific action, but it appears in some cases to have a beneficial effect in improving the appetite, promoting nutrition, and diminishing cough and secretion. Only pure beechwood creosote should be employed, and it is best given in the form of capsules after meals or mixed with milk. From 1 to 15 minims may be given three times a day. Guaiacol, one of the principal constituents of creosote, is less disagreeable in smell and taste, and may be given in the same form and doses. Carbonate of creosote, or creosotal, a syrupy liquid; carbonate of guaiacol, or duotal, benzoate of guaiacol, or benzosol, and guaiacetin,^f all three tasteless insoluble powders; valerianate of creosote or eosote, valerianate of

guaiacol or geosote, guaiacolate of piperidine, and thiocol, a tasteless and readily soluble powder, are among the most useful derivatives of creosote and guaiacol, and may be given in doses of from 5 to 15 grains or minims in cachets or capsules three times a day.

Creosote and guaiacol may be given by rectal injection dissolved in olive-oil, and they have also been used endermically by painting them over the skin.

Guaiacol, or a solution of one part of guaiacol in ten of sterilised oil, has been used hypodermically in doses of 1 to 7 minims, the injections being made into the deeper subcutaneous tissues. Creosote and guaiacol should not be given where there is a marked tendency to hæmoptysis.

Hypophosphites of lime, soda, and iron (doses 2 to 10 grains), which have a tonic action and help to improve the appetite, have found some favour. They may be given in combination in the form of the liquor hypophosphit. co. (B.P.C.), dose $\frac{1}{2}$ to 2 drachms with orange-wine.

Other remedies which have been used more or less extensively are garlic, ichthyol, oil of cloves, oil of cinnamon, and cinnamic acid. Garlic is given in cachets or chopped up with beef-tea in doses of $\frac{1}{2}$ to 2 drachms. Ichthyol is best given in doses of 20 to 60 grains in the form of capsules coated with keratin, which does not dissolve in the stomach. Oil of cloves and oil of cinnamon are given in capsules in doses of 5 to 30 minims. Cinnamic acid, an ingredient of cinnamon and Peruvian balsam, and sodium cinnamate, have been given by subcutaneous or intravenous injection, the former in a 5 per cent emulsion made with oil and yolk of egg.

Raw meat has been used extensively in France as a remedy on the recommendation of Richet and Héricourt. While, however, it has appeared to benefit in comparatively early cases, in the presence of secondary infections and toxæmia it is powerless to do good. The meat is finely minced, scraped with a knife, or thoroughly pounded in a mortar, and both the meat-pulp and the juice expressed from the fresh meat are given. In this way nearly 2 pounds of lean, raw meat are administered to the patient daily.

Formalin has been used as an inhalation, and has been administered by intravenous injection. When it is given as an inhalation, the following formula may be adopted :—

R. Formalin, ℥xij.
Sp. chlorof. ℥xxx.
Ol. menth. pip. ℥xij.

Terebene, ℥ss.

Sp. rectificat. ad ℥j.

Ft. solutio. Five to twenty minims to be inhaled through an oral inhaler for ten minutes every hour.

In this form it has proved soothing to the cough and has improved the general health. When formalin is given by intravenous injection, a solution of 1 part in 2000 parts of .7 per cent sterilised solution of sodium chloride is employed, and 50 c.c. are injected daily. No real benefit has yet been demonstrated from formalin intravenous injections. Intratracheal injections of a mixture containing izal 10 parts, menthol 2 parts, and 88 parts of glycerin have been employed and advocated by Mr. Colin Campbell (*Trans. Congress Tuberculosis*, 1901). From 2 drachms to 2 ounces are injected daily into the trachea.

Counter-irritation is useful in certain cases. If there be bronchial catarrh, rubefacient liniments, such as lin. camph. ammon. or lin. terebinth. or lin. terebinth. acetic., are useful. If there be pain, iodine paint or a mustard leaf may be applied over its seat. The pain of pleurisy may be relieved by strapping, by poulticing, or by the application of belladonna plaster.

Symptomatic treatment.—*Cough.*—The cough of chronic phthisis demands treatment when it is frequent, irritating, and useless. The remedies employed should be as simple as possible. In many cases the sipping of warm milk, or such drinks as barley-water, lemon-water, or linseed-tea, will relieve. Liquorice lozenges, Pontefract cakes, or the Brompton Hospital lozenges containing liquorice and anise are useful. Heroin hydrochloride is a useful remedy for troublesome cough, and may be given in tablets or pastilles containing gr. $\frac{1}{16}$. Such a linctus as one of the following may be given at bedtime:—

℞ Syrupi codeinæ, ℥vj
Glycerini, ℥vj.
Sp. chlorof. ℥ss.
Succ. limonis ad ℥ij.

Ft. linct. A teaspoonful occasionally.

and

℞ Tinct. opii, ℥ss.
Acid. sulph. dil. ℥80.
Oxymel. scill. ℥vj.
Sp. chlorof. ℥ss.
Syrup. limonis ad ℥ij.

Ft. linct. A teaspoonful occasionally.

Inhalations, such as 20 minims of a mixture of equal parts of creosote, spirit of menthol (20 per cent), and spirit of chloroform, or of a mixture of equal parts of terebene and spirit of chloroform dropped on the sponge of an oro-nasal respirator, help to check a useless cough. In other cases where the cough is ineffectual because the secretion is too thick and viscid, iodide of potassium with ammonium carbonate is a useful remedy. Where the secretion is profuse, balsamic remedies, such as have been recommended for chronic bronchitis with much secretion, may be tried. When fits of coughing come on after food, and are followed by vomiting, alkalies given before meals are useful. The following prescription may be employed:—

R Liquor. potass. π 80.
 Aluminis, gr. 80.
 Aquæ menth. pip. ad ℥ iv.

A dessertspoonful in a little water to be given shortly before meals.

Hæmoptysis.—On the occurrence of hæmoptysis, the patient should be kept in bed in a cool and airy room. It is highly important that the room should be kept cool, as a hot atmosphere promotes hæmorrhage. The patient should not be disturbed by anxious friends, and should not be allowed to talk. If restless and excited, he may be given a hypodermic injection of morphine (gr. $\frac{1}{4}$), and an ice-bag may be applied to the front of the chest. Oil of turpentine, 5 minims in capsule, or small doses of hamamelis or ergot may be given internally every two hours. An aperient is generally beneficial. It is desirable to limit the amount of fluid the patient gets, and the diet may suitably consist of two pints of milk, with a little bread-and-butter, custard, and calf's-foot jelly in the twenty-four hours, until all hæmorrhage has ceased. It should be seen that the patient's feet are warm, and hot-water bottles should be applied if necessary. Ice is sometimes given to suck, but its effect apart from quieting cough is probably entirely mental.

Pyrexia is generally best treated by keeping the patient at rest in bed under the best hygienic conditions, rather than by the use of any antipyretic remedies. When the patient is treated on open-air lines, as a rule the pyrexia subsides after a time. All the known antipyretic drugs have had a thorough trial, and none of them appear to have had any permanently favourable influence; but small doses of antipyrin or phenacetin may be given occasionally when the temperature is high.

Sweating at night, as has been already pointed out, is often

dependent on the fact that the air of the room is not pure, that its temperature is too high, and that the clothing is excessive. It generally ceases when the patient has plenty of fresh air and the room is kept cool. Should the sweats continue, the following remedies, to be given at bedtime, are among those which have proved useful, viz.:—Oxide of zinc, 5 grs.; extr. of belladonna, gr. $\frac{1}{2}$, or atropine, gr. $\frac{1}{100}$; extr. of nux vomica, gr. $\frac{1}{2}$, or strychnine, gr. $\frac{1}{12}$; agaricin, gr. $\frac{1}{12}$; picrotoxin, gr. $\frac{1}{60}$; and camphoric acid, 20 to 30 grs. Oxide of zinc and belladonna, either in combination or separately, are probably the most effectual.

Dyspepsia, which is a very common symptom, often requires treatment. An alkaline tonic, such as the *mistura gentiana alkalina* of the Brompton Hospital, taken before meals, often cures the dyspepsia. The formula is as follows:—

R Sod. bicarbonat. $\bar{\text{ij}}$.
 Acid. hydrocyan. dil. mxxiv .
 Infus. gent. co. ad $\bar{\text{viii}}$.

An eighth part to be taken three times a day, ten minutes before meals.

In other cases carbonate of bismuth or sulphocarbolate of soda is useful. Constipation is often present, and pills containing aloes, aloin, or cascara sagrada should be given at bedtime when required.

Diarrhœa is a complication which is apt to prove very troublesome in the later stages. It may arise from tuberculous ulceration, or from a catarrhal condition of the intestines. Preparations of bismuth, the carbonate, the subnitrate, or the subgallate, in doses of from 10 to 30 grains three times a day, substances containing tannin, such as kino and catechu, or derivatives such as tannigen and tannalbin, in doses of 5 to 10 grains, copper sulphate in small doses ($\frac{1}{4}$ to $\frac{1}{2}$ gr.), and opium, are the most useful remedies.

PULMONARY GROWTHS

The morbid anatomy of pulmonary growths has been considered at p. 98. It is rare that the lung is affected with growth without the mediastinum being also involved, except in the case of multiple secondary tumours.

Symptoms.—When the mediastinum is also the seat of growth, then the main symptoms are those produced by pressure on the important structures which are there situated. These

symptoms are fully described at p. 278. We shall here consider the symptoms which arise when the growth is limited to the lung. In some cases no characteristic symptoms are observed, and the growths, especially when they are small and secondary, are discovered unexpectedly after death. In other cases, such symptoms as pain, cough, expectoration, hæmoptysis, dyspnœa, cyanosis, anæmia, wasting, bodily weakness, and a slight degree of pyrexia have been present. Pain is sometimes the first symptom, and generally is due to involvement of the pleura or other extension of growth beyond the lung. In some cases it is slight or absent, in others it is very severe, and sometimes it is paroxysmal. It sometimes radiates to the arms, abdomen, and back, and may be accompanied with superficial tenderness. Cough is a frequent symptom resulting from coexisting bronchitis or pressure on bronchi. The expectoration is usually mucous and is not uncommonly blood-stained. In some cases it is like red-currant jelly or resembles prune juice, and occasionally large hæmorrhages have occurred. Dyspnœa is often extreme and distressing, and is probably out of proportion to the physical signs which are present. It is most marked, however, when there is pressure on the larger tubes, and paroxysmal exacerbations are then not uncommon. The rate of breathing is in some cases rapid, and in others of an asthmatic type. Cyanosis and orthopnœa are present when dyspnœa is severe. Wasting is not usually a marked feature, but the patient is generally anæmic. A moderate degree of pyrexia is not uncommon, and is sometimes accompanied with perspirations. There is usually a sense of fatigue and great bodily weakness, and the patient may suffer from attacks of syncope. Sometimes acute infiltrating carcinoma has been observed to run a course resembling acute pneumonia, with rigors, a marked degree of pyrexia, facial herpes, and rapid consolidation of the lung. Some cases run a short and acute course with fever, sweating, and rapid emaciation.

The **physical signs** are in many cases indistinguishable from those of bronchitis. In other cases there is evidence of consolidation. Local or general retraction of the lung is common when there is pressure on the bronchi, and in cases of infiltrating carcinoma. In other cases there is bulging. Respiratory movement is usually diminished or lost over the affected part, while that of the opposite lung is increased. The heart is displaced away from the affected side when there is bulging and sometimes when there is retraction. When there are scattered tumours the percussion note may be normal or even hyper-resonant. When

consolidation extends to the surface the note is commonly absolutely dull. The breath sounds vary according as the tubes remain patent or become blocked. In the former case there is bronchial breathing with increased vocal fremitus and vocal resonance; in the latter case there is partial or complete absence of breath sounds with diminished vocal fremitus and vocal resonance. Effusion into the pleura is a common complication, and the fluid is sometimes blood-stained. In some cases there is secondary affection of the glands, especially the supraclavicular, the axillary, and the subclavicular.

The prognosis of intrathoracic growths, when they can be diagnosed with certainty, is necessarily a hopeless one. The duration of the illness is variable, but it averages about twelve months from the time that symptoms first make their appearance.

Diagnosis.—The diagnosis of pulmonary growths is often a matter of difficulty. One is assisted by a history or other evidence of growth elsewhere in the body, or by signs of pressure on some of the important structures which pass through the mediastinum. The symptoms may closely resemble those of chronic bronchitis and emphysema, and suspicion may be only excited by the persistence and intensity of the dyspnœa. Sometimes the physical signs resemble pleurisy with effusion, or a growth in the lung is accompanied by effusion. The affected side in the case of growth is more often contracted than expanded. If the fluid drawn off be hæmorrhagic, or if in amount it is disproportionate to the extent of dulness present, it is in favour of growth. Other cases suggest chronic pulmonary tuberculosis, especially the fibroid type. The absence of tubercle bacilli in the expectoration is an important point. The result of the tuberculin test, applied as described at p. 229, would be useful, but sometimes a reaction has been obtained in cases of carcinoma, and it is possible that both tubercle and growth may be present. Physical signs of consolidation at both apices would be in favour of phthisis. In the case of growth the dulness is usually more intense, and more often accompanied by suppression or marked diminution of the breath sounds and of vocal fremitus and resonance, than in the case of phthisis. An extension of the dulness across the middle line would be in favour of growth. Other points are mentioned in the article on mediastinal growths.

Treatment.—The treatment of pulmonary growths can necessarily only be palliative and symptomatic. Life may be prolonged by fresh air and good food. Pain can be relieved by morphine,

and dyspnœa by oxygen inhalations, or by some of the measures useful in spasmodic asthma.

HYDATIDS OF THE LUNG

Symptoms.—Even large tumours may exist for a long time without giving rise to any serious symptoms, and are compatible with good health. The period previous to rupture is one of physical signs rather than of symptoms, and the signs of tumour may be discovered quite accidentally, as, for instance, during examination for life insurance. The patient, however, may suffer from cough, hæmoptysis, pain, or dyspnœa. The cough may be dry and hacking, or attended with mucous or muco-purulent expectoration. Sometimes it is paroxysmal and laryngeal in character. The sputa are not uncommonly blood-stained, and when rupture is impending, there may be a considerable hæmorrhage. When the hydatid involves the pleura, severe and persistent pain is likely to be present, seated in the tumour, or referred to the shoulder or epigastrium. In other cases there may be a sense of weight or discomfort, but no actual pain. Dyspnœa is usually slight, except when the hydatid is large, and even then it is rarely severe. While the cyst is unruptured the general health as a rule remains good, and the temperature is normal.

Physical signs.—There may be bulging of the chest-wall on the affected side, either generally or partially, the swelling in the latter case being somewhat globular. The intercostal spaces may be distended, obliterated, or protruding. The respiratory movements on the affected side may be diminished or even abolished. The heart's impulse is usually displaced away from the affected part. If the cyst be superficial, absolute dullness will be observed over it, and on palpation a sense of elastic resistance may be noticed or distinct fluctuation may be obtained. The breath sounds are suppressed over the dull area, and vocal fremitus and resonance are diminished or absent. Pleuritic friction may be present, and is a valuable sign when audible over an area giving the signs of fluid. Examination by means of the Röntgen rays may show decisively the presence of a rounded tumour.

The cyst ruptures spontaneously in about half of the recorded cases. The rupture usually occurs into a bronchus, giving rise to sudden pain, intense dyspnœa, and the expectoration of a large amount of watery, blood-stained fluid, sometimes containing

daughter cysts. The lungs may be so completely flooded with fluid that death rapidly ensues. Profuse hæmoptysis is not uncommon at the time of rupture. If the patient be not immediately carried off, he may continue expectorating daughter cysts for days, or even weeks. When the cyst ruptures into the pleura, hydro-pneumothorax occurs, which frequently proves fatal.

After rupture the interior of the cyst may suppurate, and symptoms of pulmonary abscess develop, as shown by emaciation, hectic temperature, profuse, purulent, and often fœtid expectoration, severe cough, and occasional hæmoptysis. The physical signs are those of a cavity.

Prognosis.—Hydatids of the lung may last for years, but left to themselves they are a source of constant danger to the patients affected. Spontaneous rupture may take place at any time and prove directly fatal by flooding the lungs. Death may also occur from pneumothorax, hæmorrhage, rupture into the pericardium, pulmonary abscesses, or pneumonia. Surgical treatment at an early stage greatly improves the prospect of recovery.

Diagnosis.—The diagnosis of pulmonary hydatids is a matter of considerable difficulty. The conditions for which hydatids are most likely to be mistaken are pulmonary tuberculosis, pleural effusions, and other intrathoracic tumours. The situation and form of the area of dulness, and the combination of physical signs and symptoms already mentioned, will help in arriving at a diagnosis, and a Röntgen-ray examination should be made. Exploratory puncture should not be performed without preparations for freely opening up the cyst at once, should one be found. In the case of hydatid the general health is better maintained than in a case of phthisis with the involvement of a similar extent of lung. Moreover, the symptoms are not those of toxæmia, but result from the mechanical effects of pressure and interference with the amount of breathing space. The limitation of the disease to one lung and the absence of signs of excavation or softening are other important points of distinction from phthisis. The situation and form of the area of dulness will usually serve to distinguish a hydatid from a pleural effusion, which, even when circumscribed, has seldom the rounded and somewhat globular shape of the hydatid. The differentiation from other intra-thoracic tumours depends on the absence of signs of mediastinal pressure, on the presence of fluctuation on the rounded form, and on the absence of vocal fremitus and resonance and of the breath sounds over the dull area.

Sometimes there is a difficulty in distinguishing between cysts in

the upper part of the liver and those in the lower part of the right lung. The outline of the dulness and its continuity with the liver dulness, and the association with liver enlargement and displacement in the case of hepatic hydatid, will help. When the lung is perforated by a liver cyst the sputum may be tinged with bile.

Treatment.—Whenever a pulmonary hydatid is diagnosed, the treatment must be purely surgical. The cyst should be freely incised, which is the safest and most satisfactory mode of treatment. Although many recoveries have occurred after simple tapping, this proceeding is, we consider, unjustifiable, as it is attended by considerable risk to life from the danger of the cyst rupturing into a bronchus as the result of tapping. When suppuration has occurred, incision and free external drainage should be carried out, as in the case of ordinary abscess of the lung.

SYPHILIS OF TRACHEA, BRONCHI, AND LUNGS

Symptoms.—The chief symptoms of syphilis of the trachea are dyspnœa and inspiratory stridor. The degree of dyspnœa will depend on the amount of obstruction of the trachea. The dyspnœa is sometimes paroxysmal in character, resembling that produced by pressure on the trachea from without by aneurysm or growth. Cough and expectoration generally precede or accompany the dyspnœa. The voice may be weak, hoarse, or croupy. Examination by the laryngoscope may show the presence of a gumma or cicatricial contraction. Syphilitic lesions of the bronchi are usually associated with syphilis of the trachea. The symptoms of syphilitic stenosis of both bronchi will resemble those of stenosis of the trachea. Acute symptoms may show themselves after a long period of latency as the result of accumulation of secretion resulting from an accidental bronchial catarrh.

The symptoms of syphilis of the lungs are not characteristic. In the early stages they are those of bronchial catarrh, with cough and shortness of breath, which become more marked in the later stages. The expectoration, at first mucoid and small in amount, becomes later more profuse and muco-purulent or purulent, and may be offensive and contain fragments of pulmonary tissue. Hæmoptysis, generally slight but sometimes profuse, is not infrequent. Dyspnœa may be paroxysmal, as when the trachea is involved, and pain in the side sometimes accompanies the dyspnœa. The temperature is usually normal, but fever has been recorded in

some cases. Although sometimes the general condition is good, anæmia, emaciation, and marasmus are frequently present. The physical signs are those of consolidation and excavation, most likely occupying the middle portion of the lungs.

The **diagnosis** of syphilis of the trachea must be arrived at by the absence of other causes of obstruction, and a history of syphilis or evidences of it elsewhere. The diagnosis of syphilis of the lungs is almost impossible, but with a history of syphilis, or with evidences of syphilitic lesions in other parts of the body, syphilis may be suspected in cases of pulmonary disease, with symptoms such as have been described, in which phthisis can be excluded.

Prognosis.—Tracheal gummata may be entirely cured by treatment, but cicatricial stenosis cannot be removed. In pulmonary syphilis the prognosis is good, unless the lesions are associated with grave disease of other viscera, provided active anti-syphilitic treatment be adopted. Albuminuria is an unfavourable symptom.

Treatment.—Iodide of potassium combined with small doses of belladonna or atropine should be given in all cases of suspected syphilitic disease of the lungs or air-passages. In syphilitic stenosis of the trachea, tracheotomy is indicated, provided an opening can be made below the situation of the lesion.

HECTOR MACKENZIE.

DISEASES OF THE PLEURA

GENERAL ETIOLOGY

The pleuræ, from their close connection with the thoracic walls, and also with the lungs which they envelop, are manifestly liable to become involved in lesions of those structures, and such pleuritic affections may be regarded as secondary. These membranes are also the seat of primary diseases, determined in great measure by the communications which exist between the pleural cavities and the pulmonary, parietal, and diaphragmatic lymphatics, and indirectly by means of the last-named by the continuity which is established with the cavity of the peritoneum. Some maladies of this class are doubtless determined by blood states, whilst the determining cause of others, *e.g.* some new growths, is at present unknown.

Neither *age* nor *sex* appears to exert any special effect on the incidence of pleural affections. So far as both of these conditions favour exposure to the exciting causes of disease, or modify the nutrition and resisting power of the tissues, they are to be considered as predisposing; and thus may be explained the far greater frequency with which the effusions of acute inflammation of these membranes assume a purulent character in children, and also that acute pleurisy occurs more commonly in early and young adult life. It has been noticed that those cases of pneumothorax occurring in the apparently healthy are almost invariably males.

Trauma.—Injury of the chest-wall, such as fractured ribs, stabs, or other wounds, are most likely to injure the pleura, and possibly also the lung, and as a result thereof a localised pleurisy may be set up; or, should the wound have admitted septic matter, the resulting inflammation may be more general, and perhaps even purulent. In some cases injury may be followed by a tuberculous pleurisy; the spread of the tubercle from the lung being favoured by the damage done to the serous membrane. Should a vessel of any size be severed, an escape of blood into the pleural cavity—hæmothorax—may follow, and very rarely this may be complicated with air—hæmopneumothorax. Air alone—pneumothorax—is a not uncommon result of a wound of the chest-wall communicating with the pleura; and this accident has occasionally

happened in thoracentesis, and as a consequence of tracheotomy. This condition, however, is a far more frequent consequence of the rupture of an air-vesicle in the lung, the result of tubercle, malignant disease, hydatid, abscess, or gangrene of the organ. The possibility of the rupture of a vesicle taking place under the influence of great strain or from violent coughing, as in whooping-cough, must be borne in mind. Emphysema is by no means so important a factor in producing pneumothorax as was at one time supposed; only seldom is the evidence conclusive of a burst emphysematous bulla being the cause.

Similarly pleurisy, pneumothorax, hæmothorax, or empyema may be caused by the rupture of the œsophagus, of a thoracic aneurysm, or of an hepatic or a subdiaphragmatic abscess, or the perforation of a gastric ulcer into the pleural cavity, or of the escape of the contents of an hydatid cyst into the same.

At one time *cold* was regarded as a frequent cause of pleurisy, but increasing evidence is being accumulated in favour of the view that pleurisy is generally a secondary condition, and that in the cases of so-called idiopathic origin, or in those due to *chill*, the real cause of the disease has been overlooked, such effect as cold has being in the direction of rendering the body more vulnerable.

Infection.—The probability is that all cases of pleurisy are due to the irritation of certain micro-organisms or their products, and that the conditions already enumerated, which have hitherto been regarded as the direct causes of pleurisy, act indirectly by lowering the resistance of the individual and thus allow infection to take place.

Of late much attention has been directed to the bacteriology of pleurisy, and the following varieties have been described:—

1. *Pneumococcus pleurisy* (sero-fibrinous and purulent).—This variety may be primary, but it is more often secondary to pneumonia, and it sometimes occurs in connection with otitis media. It is the usual cause of empyema in childhood.

2. *Streptococcus pleurisy*.—This is the common form of empyema in adults. It may be primary, but more frequently it is secondary to pneumonia, influenza, scarlet fever, and septic diseases.

3. *Purulent tuberculous pleurisy*.—This is usually either the consequence of pulmonary tuberculosis, or of tuberculous bronchial glands. A purulent pleural effusion may occur in a tuberculous subject, as a result of pneumococcic or streptococcic infection and therefore independently of tuberculosis. Though tuberculosis is rightly regarded as the commonest cause of pleurisy with serous effusion, tubercle bacilli are seldom found in the fluid.

4. *Putrid pleurisy*.—This form is always purulent, and it owes its putridity to saprogenic organisms. Streptococci and staphylococci may also be present. Staphylococci are met with in association with tuberculous and other forms of pleurisy, but it is doubtful whether the staphylococcus alone gives rise to pleurisy.

Finally, cases of pleurisy due to the presence of the gonococcus and of Eberth's bacillus have been recorded.

Pleurisy not infrequently arises in association with septic forms of peritonitis, such as occur in the puerperal state, the infection being conveyed by the lymph-canals between the diaphragm and pleura. There is a general tendency for the several serous cavities to be simultaneously affected.

As complications of, or secondary to, other diseases, affections of the pleura are of constant occurrence. The primary affection may be (*a*) of the lungs, (*b*) of the chest-wall, or (*c*) remote from the respiratory organs.

(*a*) *Of the lungs*.—Most pulmonary diseases are sooner or later liable to lead to involvement of the pleura, and very frequently determine some form of pleurisy. Thus pulmonary tuberculosis, pneumonia, broncho-pneumonia, malignant disease, abscess, and gangrene are usually so complicated. The pleurisy is often of the dry variety, but in children a large proportion of the empyemas is due to pneumonia, and nearly 70 per cent of cases of apparently simple pleurisy with serous effusion owe their origin to tubercle. Acute purulent tuberculous pleurisy is, however, an exceedingly rare condition.

(*b*) *Of the thoracic parietes*.—Abscess, new growth, etc., in this situation, may extend to the pleura, and in like manner erysipelas of the chest-walls may lead to pleurisy.

(*c*) Reference has already been made to those pleural affections which are due to rupture of an abscess or aneurysm in adjacent regions, and to the perforation into the pleural cavity of a gastric ulcer. Pleurisy is also an occasional complication of acute infective diseases, especially measles, scarlet fever, influenza, and acute rheumatism, and it may also occur in the course of Bright's disease. Sanguineous effusions into the pleural sacs are met with in altered blood states, such as purpura, scurvy, and hæmophilia.

MORBID ANATOMY AND PATHOLOGY

In PLEURISY, as in inflammation of other serous membranes, the vessels of the pleura become dilated, and the surface loses its

smoothness and polish from the exudation of leucocytes and fibrin. As a part of the inflammatory process an effusion takes place, which varies much in quantity and character. Thus it may be (1) *fibrinous*, either very scanty as occurs in the so-called "dry pleurisy," when the pleural surfaces are covered with an adhesive lymph, which presents a shaggy or honey-combed appearance from the rubbing caused by the respiratory movement, or it may be more abundant as in cases of pleurisy accompanying pneumonia. Under favourable circumstances the exudation may in time be completely absorbed, or, as probably oftener occurs, the lymph becomes organised, and fibrous adhesions are formed between the surfaces of the pleura, which is thickened, with some fibrosis extending into the pulmonary tissue.

(2) *Sero-fibrinous exudation*. — The very abundant effusions, amounting to several pints (as many as fourteen have been recorded), are of this character. The fluid, which is of a variable specific gravity, from 1005 to 1020, is usually of a clear straw colour, or may be slightly turbid from cell elements, leucocytes, pus-cells, and red blood-corpuscles. Fibrinous coagula are found in the fluid *in situ*, or they may form after withdrawal from the chest. Cholesterin, sugar, urea, and uric acid have been met with in such effusions. The surfaces of the pleura in these cases may be covered with a thick layer of fibrinous shreds, enclosing the more fluid exudation in an incomplete meshwork, which may be ultimately absorbed.

(3) *Sero-purulent or purulent empyema*. — This is commonly the result of pneumococcic or streptococcic infection, the effusion consisting of more or less pure pus with flakes of lymph and fibrin. Should putrefactive organisms be present, as from gangrene or a septic infarct of the lung, or from communication with an abdominal abscess, the effusion may become putrid and of fœtid odour.

The detection of the tuberculous nature of a pleural effusion is a matter of great difficulty. The tubercle bacillus is usually absent from the effusion, so that staining, even after centrifugalisation, affords no assistance, nor have attempts at cultivation been much more successful. Injection of the pleural fluid into the peritoneal cavity of guinea-pigs gives fairly reliable results, but it is necessary to wait four or five weeks to obtain them. Tuberculin injections (p. 228) are said to give very satisfactory results and to be free from risk, but up to the present time they have not been looked upon favourably in this country. A new method of examination—cytodiagnosis—promises to be of great use. It depends upon the nature of the cells found on microscopical examination in sero-fibrinous effusions. Fluid,

withdrawn by a sterilised syringe, is defibrinated and centrifugalised. The deposit thus obtained is spread on a slide, and stained with eosin and hæmatein. The following cells may be recognised:—red blood-corpuscle, flakes or masses of endothelium from the pleural surface, and the ordinary white blood-cells, lymphocytes, polymorphonuclear neutrophiles, or occasionally eosinophiles. By this method of examination, Widal and Ravaut have classified pleurisies into three groups, according to the prevailing variety of cell present: in tuberculous cases, lymphocytes; in other infections, polymorphonuclear neutrophiles; in mechanical, *i.e.* non-infectious (cardiac, renal, etc.), endothelial flakes.

Fluid in the pleura, whatever its character, is said to be loculated when it is shut off by adhesions from the general cavity of the serous sac.

As a result of the presence of the fluid the lung may be much compressed, and in cases of large effusion it may be reduced to less than a sixth of its normal size, being displaced upwards, inwards, and backwards, so that it comes to occupy the upper part of the thoracic cavity close alongside the spinal column. Owing to the formation of membranous exudations on the visceral surface of the pleura, expansion of the lung may be prevented if the process of absorption be slow or paracentesis be performed late.

In the chronic form of pleurisy the fluid may be entirely absorbed, but the lung is left more or less collapsed. The pleura is greatly thickened, and there is an increased growth of fibrous tissue, extending from the pleura to the interlobular connective tissue. In well-marked cases the lung is solid and airless. To this condition the term “pleurogenic cirrhosis” has been applied.

Three varieties of PNEUMOTHORAX have been described, viz. open, closed, and valvular. Open pneumothorax occurs in cases of penetrating wounds of the chest, when there is a free communication between the external air and the pleural cavity. Closed pneumothorax is the term applied to cases in which the opening into the pleural cavity has become sealed by adhesive inflammation. Owing to the absorption of air which commonly takes place, there may be negative pressure in the pleural cavity. In valvular pneumothorax a piece of lymph or portion of the pleural membrane may act as a valve and allow air to enter at inspiration, but not to escape during expiration; in a case of this kind there will be positive intrapleural pressure, and the physical signs are consequently very marked. In cases of death following complete pneumothorax, on opening the chest, air will escape with sufficient

force to extinguish a candle, if there have been positive intra-pleural pressure, and the lung will be found collapsed. The air in the pleural cavity consists chiefly of nitrogen with some carbonic anhydride and a smaller amount of oxygen; if putrefactive changes have taken place, sulphuretted hydrogen will be present. The existence of tuberculosis, gangrene, abscess, or other cause of the pneumothorax is generally easily recognised, but it may be difficult in exceptional cases to determine the precise cause of the rupture. A localised pneumothorax is probably often overlooked at the necropsy. Unless death takes place very speedily pneumothorax is usually accompanied by a secondary pleurisy, and the effusions met with are, roughly speaking, serous, sero-purulent, and purulent in about an equal proportion of cases.

HYDROTHORAX occurs as a part of general dropsy, and is most commonly due to chronic valvular disease of the heart or to chronic Bright's disease. It also occurs in connection with cirrhosis of the liver and in cachectic conditions. It may arise from the pressure of an intra-thoracic tumour on the azygos veins. Unless due, as in the last instance, to a local lesion, hydrothorax is generally, but not invariably, bilateral, though not always to an equal extent on the two sides. The effusion met with in cases of hydrothorax is a clear serous fluid, and differs from that poured out in inflammatory conditions in containing only a few flakes of fibrin; there is consequently no tendency for the lung to be bound down by adhesions, and it is simply collapsed.

HÆMOTHORAX is an extravasation of blood into the pleural cavity. It may result from an injury, most frequently the puncture of an intercostal artery or laceration of the lung by a fractured rib. Very copious effusion of blood occurs when a thoracic aneurysm ruptures into the pleural cavity. Cancer of the lung or pleura is sometimes accompanied by free hæmorrhage into the pleural cavity. A blood-stained exudation is met with in some cases of tuberculosis of the lung, in purpura, scurvy, leuchæmia, cirrhosis of the liver, granular kidneys, and other diseases attended with destructive changes in the blood, also as a sequence of degenerate vessels.

The condition of the blood varies, sometimes remaining fluid, but in traumatic cases it is usually clotted.

CHYLOTHORAX is an effusion of a chylous fluid into the pleural cavity. It is of very rare occurrence, and is due either to traumatic rupture or to obstruction of the thoracic duct.

NEW GROWTHS.—Of the innocent tumours, fibroma, osteoma, and lipoma have been met with in the pleural cavity, but on account

of their rarity and the absence of symptoms it is unnecessary to do more than mention them.

Carcinoma and sarcoma both occur in the pleura, usually in the form of secondary deposits, the pleura being implicated either by metastasis or by extension of the disease from adjacent parts. Cancer of the breast is the most common cause of malignant disease of the pleura.

Primary sarcoma or carcinoma of the pleura is a very rare affection. Hydatid tumour of the pleura is very seldom met with in this country, and when it does occur it is usually secondary to hydatid of the liver.

PLEURISY

Symptoms.—The symptoms of ACUTE PLEURISY occasionally come on with great suddenness, being ushered in by rigors; more frequently, however, the patient complains merely of chilliness. The symptom on which his attention is concentrated is a sharp, catching, stitch-like pain in the side of the chest, “as actively annoying and as petulantly resented as a toothache.” In cases which come on more insidiously, pain is less marked and may be felt only on taking a deep inspiration. Coughing is not a prominent symptom; it is generally dry, or it may be accompanied with a little mucous expectoration. The cough is restrained as much as possible on account of the great pain experienced by the patient when he does cough. The respirations are increased in frequency, so as to make up for their want of depth; a full inspiration greatly intensifies the pain—the breathing is therefore short and of a gasping character, and is of the abdominal type. At the commencement of the attack the patient is unable to lie on the affected side. The pulse is frequent, and usually rather full and bounding; its frequency increases, more or less, in proportion with the frequency of the respiration. The temperature is not as a rule very high, 100° to 103° F. being about the usual range. The skin may be moist, and it has not the pungent heat met with in cases of pneumonia. In pleurisy complicating pneumonia the symptoms of the more superficial and less severe disease are thrown into the shade by the graver malady. Together with the increase of temperature, there are the usual symptoms of the febrile state, such as anorexia, thirst, furred tongue, constipation, and scanty, high-coloured urine; except for the increased specific gravity consequent on the diminished output of

water there is usually no other change in the constituents of the urine, and they retain their normal relative proportion. Occasionally, however, a trace of albumen is present, which may be referred to the febrile state of the patient.

The symptoms may be arrested at this stage, and the patient gradually return to his usual health without any fresh development. More commonly, however, in acute pleurisy the dry stage is followed by the stage of effusion, and then a fresh set of symptoms manifest themselves. The patient loses the acute pain, which was so prominent a feature at the onset, but now complains of a feeling of weight and fulness in the affected side. He can no longer lie on the sound side, but lies towards the affected side, but generally not on it; dyspnœa is usually a marked symptom, and extreme orthopnœa is sometimes seen. The complexion is often dusky on account of the interference with the aeration of the blood. If cough be present it is not of the painful character met with in the first stage. The temperature is not characteristic; in some cases there may be considerable elevation, and it may remain fairly steady between 103° and 104° F., as a rule, however, it is lower than in the first stage; high evening temperature with morning remission points, as we shall see later on, to empyema. The general symptoms of this stage are the same as those occurring in the first stage. If, as usually happens, the case runs a favourable course, the temperature falls by lysis, the respirations and pulse decrease in frequency, and the patient is gradually restored to health; this process being much accelerated by paracentesis in suitable cases. A leucocytosis is usually present in pleural effusions.

Physical Signs.—In the first stage, on inspection there will be noticed diminished movement on the affected side. On palpation friction fremitus can generally be detected. The percussion note is not materially altered. On auscultation there is diminished entry of air and the respiratory rhythm is irregular and jerky. The characteristic sign of pleurisy is the rough to-and-fro friction sound. At the commencement a rub may be audible only at the end of inspiration, and in diaphragmatic pleurisy no friction may be detected. The most common situation of a friction sound is in the infra-axillary region, or towards the angle of the scapula. If the disease goes on to the second stage, the signs of effusion are present. Should the effusion be extensive the affected side will tend to become rounder and there will be almost entire absence of respiratory movement on the side; the intercostal spaces may be obliterated, but bulging usually occurs only when an empyema is about

to burst. If the effusion have existed for some time, especially in children, there may be retraction of the side. In right-sided effusions the heart's impulse will be found outside the left nipple, and in left-sided effusions the impulse may sometimes be recognised to the right of the sternum. Displacement of the heart has been noticed as early as the fourth day, and it may be the result of only a moderate effusion. Depression of the diaphragm pushes down the liver or spleen ; the latter condition is rare. Displacement of viscera takes place to a much less extent in the pleurisy of children than in adults, owing to the yielding nature of their thoracic walls. Vocal fremitus is usually quite absent. On percussion there will be found an entire absence of resonance ; in front there is usually an abrupt line of demarcation between the dull and the resonant parts of the chest wall ; posteriorly, however, the dulness gradually passes into a more resonant note. Where there is a large quantity of fluid in the pleural cavity the peculiar sub-tympanitic note, to which the term of Skodaic resonance has been applied, may sometimes be obtained in the infra-clavicular region. As a rule change of position has but little, if any, influence on the line of dulness, but occasionally the area of dulness gradually shifts if the patient alter his position. The auscultatory signs of pleural effusion vary very much. The most characteristic is entire absence of respiratory murmur. In children, breath sounds can often be heard distinctly through the fluid. In some cases feeble bronchial breathing, or even breathing of a tubular character, can be heard over the compressed lung along the spine. Over the unaffected lung puerile breathing is usually heard. Vocal resonance is commonly lost, but in cases where there is a thin layer of fluid with compressed lung behind, a peculiar bleating sound, to which the term "œgophony" is applied, may be heard at the upper limit of the fluid. In the natural process of cure, as the fluid is absorbed the movement of the chest gradually increases, and there is a return of the friction sound for a time. The dulness gradually disappears from above downwards, the last part to remain dull is generally the infra-axillary region. The vesicular breathing returns slowly, at first it is weak and distant, then somewhat harsh, and some time elapses before it resumes its normal character. The vocal resonance may remain œgophonic until the fluid clears up, or becomes bronchophonic before the normal vocal resonance is restored. Where recovery is incomplete the thickened pleura causes impairment of resonance and diminished breathing over the affected area.

Diaphragmatic pleurisy is at the commencement oftentimes

very difficult to diagnose. The patient may be thought to be suffering from acute peritonitis, such as arises in connection with appendicitis. Owing to the fixation of the diaphragm there is hardly any abdominal movement, and respiration is confined to the upper part of the thorax. The patient usually has an anxious expression similar to that seen in severe abdominal affections; he complains of intense pain along the line of attachment of the diaphragm and in the abdomen, and there is acute tenderness. Hiccough and vomiting may occur. Friction sounds are rarely audible, and the symptoms usually abate when effusion takes place. Post-mortem evidences of pleurisy in this situation, such as adhesions, are however often found without any history of symptoms referable thereto. The best marked examples of this condition are seen in cases of pleuro-pneumonia.

EMPHYEMA.—This is the term applied to effusions in the chest of a purulent nature, they may be so from the commencement, or effusions originally serous may become purulent. In the days before the antiseptic method of treatment, the use of dirty aspirators occasionally led to this result. In childhood, owing to rapid cell proliferation, effusions are prone to become purulent. There are no diagnostic symptoms of empyema. As a rule the temperature is of the hectic type, but it must be remembered that empyemata sometimes run their course with little or no elevation of temperature, and on the other hand that cases of simple pleural effusion are occasionally attended by remittent fever. The aspect of the patient is sometimes suggestive, a waxy, earthy complexion being frequently seen, and clubbing of the finger-ends being also present. A similar want of diagnostic characteristics is met with in the physical signs. Bulging of the interspaces is more marked in empyema than in other forms of effusion. The only sure method of diagnosis is to puncture with an exploring syringe.

It has been shown that when the pneumococcus (Fraenkel's) invades the pleura it produces the same constitutional symptoms as if the lung were involved; the symptoms being due to the absorption of the toxins produced by the pneumococcus. Consequently if in any given case the signs of pneumonia are atypical, and particularly if the physical signs do not clear up readily, the chest should be explored. In children there is especial need to be on the look out for empyemata, as they often come on quite insidiously with wasting, cough, and night sweats.

Occasionally pulsation may be detected over a pleural effusion; this condition is practically confined to empyemata of long standing.

Two forms have been described. When the pulsation is *intra-pleural* it is usually only to be felt by the hand, it is hardly ever visible and it is generally widespread. *Extra-pleural* pulsation is met with only in empyema, and is limited to the external tumour. These pulsating tumours may occur at any part of the chest, but most usually they are met with near the heart. The writer saw a good example in a case in which an empyema recurred after the removal of portions of two ribs; the pulsation was very marked over the bulging through the opening between the ribs. When the situation of the pulsating empyema is in the neighbourhood of the heart, the difficulty in the diagnosis may be very considerable. The presence of a murmur would exclude an empyema, but as is well known many aneurysms occur without a murmur. Puncturing with a small exploring syringe would probably do no harm, if the tumour were aneurysmal, and the withdrawal of pus would of course be conclusive evidence of an empyema.

If left untreated, empyemata may burst externally, or may rupture into the lung and be got rid of by coughing, though cases of suffocation from the sudden entrance of a large quantity of pus into the bronchi have been recorded. The usual situation for spontaneous perforation is the third to the fifth intercostal spaces, most commonly the fifth, except in children in whom it is higher, and nearly always in the anterior, or cartilaginous part of these interspaces. If the pus be not discharged in one of these two ways or does not burrow in other directions, it may gradually dry up, the more fluid portions being absorbed, and a cheesy mass left behind, which may eventually become calcified.

DRY PLEURISY.—The term “dry pleurisy” has been applied to those cases of inflammation of the pleura in which the exudation is scanty and of a fibrinous nature. The symptoms and physical signs are those of the first stage of acute pleurisy, though as a rule the pain is not very severe. As the name implies, there is no effusion of fluid into the pleural cavity.

CHRONIC PLEURISY.—Two varieties have been described. In the one there is effusion, and the other is simply a chronic form of “dry pleurisy.” In chronic pleurisy, when effusion occurs, it is usually latent, *i.e.* there are no pronounced symptoms at the onset to attract attention to the chest, so that it is usually only the gradually increasing dyspnoea which leads to an examination of the chest and detection of the fluid. The probability is that, as in the acute form, the majority of these cases are tuberculous in origin. The fingers are usually markedly clubbed. The physical signs differ

in no respect from those met with in the effusion of acute pleurisy.

In chronic "dry pleurisy" there may be shortness of breath and cough, with a feeling of oppression and tightness about the chest. Changes of temperature will sometimes cause pain of a stitch-like character over the affected lung.

On inspection the affected side of the chest will be found retracted and with defective mobility, and sometimes the heart may be displaced. Vocal fremitus is usually diminished, but friction fremitus is often distinctly felt. The resonance on percussion is generally impaired. At places the chest may be quite dull, especially at the base, whereas higher up the resonance may be of a woodeny or boxy character. On auscultation the respiratory murmur may be feeble or absent, or have a bronchial character. Dry creaking or rubbing sounds are usually to be recognised. Vocal resonance is diminished or lost.

Diagnosis.—In the first stage pleurisy requires to be distinguished from other kinds of pain affecting the chest-wall; pleurodynia is a gouty or rheumatic myalgia, affecting the intercostal muscles, the pain is increased by movement, but there is no rise of temperature and no friction sound can be heard; the pain of neuralgia is referred to the distribution of the nerves implicated; the pain which precedes shingles is sometimes mistaken for the onset of pleurisy—curiously enough, shingles is occasionally accompanied by a rub as though there were an exudation on the pleura, similar to that which is seen on the skin.

Pleural effusion has to be differentiated from a thickened pleura, the consolidation due to pneumonic or tuberculous infiltration, new growths and aneurysm. Collapse of the lung due to a foreign body in a main bronchus sometimes simulates effusion. On the right side tumours of the liver, especially hepatic abscess or hydatid, may give rise to difficulties in diagnosis. Since the introduction of the exploring needle as a routine means of diagnosis, most of the former difficulties have disappeared. One of the most valuable results of the use of the needle is the determination of the nature of the fluid in any given case, and as its use is practically unattended by risk, the chest should be explored if there be the slightest reason to suspect a purulent effusion. It must be remembered, however, that its positive value is greater than its negative: *i.e.* the failure to draw off fluid by the needle does not absolutely exclude its presence; this is particularly the case where the fluid is a thick pus. In quite a considerable number of cases the with-

drawal of a small quantity of fluid for diagnostic purposes has been followed by the rapid absorption of the remainder. The needle is usually sufficient to exclude a thickened pleura, and measurement of the chest will generally show some contraction of the affected side.

In consolidation from pneumonia and tubercle there are increased vocal fremitus, bronchial or tubular breathing, and bronchophony, whereas in pleural effusion these signs are almost always absent. Moreover, the dulness due to consolidation does not transgress the median line as in large pleural effusions. A new growth in the pleural cavity may give to perfection all the physical signs of an effusion. The writer has recorded a case of rapidly-growing sarcoma in which the mode of onset as well as the physical signs closely resembled those of a pleural effusion.

The persistence of an evening rise of temperature in a child who has had an acute pulmonary affection, or whenever the health of a child is unsatisfactory, if it sweats at night, loses its appetite and wastes even though there be nothing pointing to the chest, should lead to a careful physical examination of the chest, and any area of dulness should be explored for pus. A drachm of pus will suffice to keep the temperature up and the child in an unsatisfactory state of health. In tumours of the liver projecting into the thoracic cavity, the upper line of dulness is not so level as in pleural effusions, and the dulness is usually more marked anteriorly and in the axillary region, fair resonance being obtainable along the spine.

In children, especially in cases of diaphragmatic pleurisy, at the commencement all the symptoms may be referred to the abdominal cavity, so that unless a careful physical examination be made the existence of the thoracic mischief may be overlooked. Again, in children of a neurotic temperament, the occurrence of convulsions, followed by headache and fever, may draw the attention of the practitioner away from the chest, and tuberculous meningitis may be diagnosed instead of pleurisy.

The diagnosis, however, is not to be regarded as complete with the detection of friction or of fluid; but the underlying causal condition, tuberculous or otherwise, must be sought for, and if possible established.

Prognosis.—As has been already stated, recovery is the rule in acute pleurisy; this may be complete, the effusion being rapidly absorbed or removed by paracentesis before adhesions have had time to form and to bind down the lung. In other cases the patient

recovers with a more or less damaged lung, owing to the thickened pleura preventing its full expansion. Occasionally a serous effusion becomes purulent, this accident may happen if paracentesis is not performed aseptically. Death but rarely occurs from acute pleurisy, when it does happen, it is due to sudden cardiac failure owing to the interference with the action of the heart from a large effusion, or to respiratory failure in cases of double pleurisy, or where in one-sided pleurisy the lung on the unaffected side is attacked with bronchitis. It is extremely dangerous for the patient to make any exertion when the heart is much displaced, getting out of bed, for instance, has been followed by death from syncope.

Though the immediate prognosis of acute pleurisy is usually good, it has been shown by carefully following up cases of simple pleural effusion without a suspicion of tuberculosis at the time, that upwards of forty per cent die of pulmonary tuberculosis within five years from the original attack of pleurisy. This is a clear indication that in pleurisy the probability of a latent tuberculosis should be borne in mind; hence the prognosis, in respect to the future, should be guarded, especially in patients with an hereditary history of phthisis.

As regards empyema it is to be noted that when it is caused by the pneumococcus the prognosis is favourable, and in these cases a cure may be effected by a single aspiration. The tendency to death in cases of untreated empyema is by asthenia. Unlike what occurs in serous effusion, empyema is very rarely followed by pulmonary tuberculosis. The possibility of the supervention of cerebral abscess should be remembered.

Occasionally spontaneous cure takes place, when this occurs it is usually due to rupture through the lung.

Treatment.—In the severer forms of acute pleurisy the patient should be put to bed in a well-ventilated room kept at a temperature of between 60° and 63° F. His diet should be light, consisting of milk, beef-tea, custard, and milk puddings. If the bowels are confined it is well to begin the treatment with a dose of calomel, three to five grains. The symptom which calls for immediate treatment is the pain in the side. This is frequently relieved by the application of a large linseed meal poultice with a little mustard. If the patient be plethoric, four or six leeches, applied over the painful region, will prove beneficial. In some cases, especially in neurotic subjects, the pain is so excessive that an hypodermic injection of morphine gr. $\frac{1}{6}$ will be necessary to give relief. Should the pain persist in spite of these measures, great benefit often

results from strapping the side in order to render it immobile. Salicylate of sodium in doses of from ten to twenty grains given every four hours will usually have a good effect in bringing down the temperature and checking the inflammatory process; but it is of no use in purulent effusions. An occasional dose of five grains of phenacetin with two grains of the citrate of caffein will relieve headache and the discomfort connected with the febrile state. If the salicylate disagree, a simple diaphoretic mixture of the solution of acetate of ammonium and spirit of nitrous ether in camphor water may be ordered. The cough which accompanies pleurisy belongs to the category of useless coughs, and should therefore be checked if possible; this can be done by small doses of the liquor morphinæ hydrochloridi (℥ 5 to 10) occasionally, or the following linctus may be employed, equal parts of compound tincture of camphor, oxymel of squills, and syrup of tolu, a teaspoonful for a dose. In the milder cases of pleurisy it may be hardly necessary to keep the patient absolutely in bed, he should, however, be confined to the bed-room, and kept as quiet as possible. When there is but little constitutional disturbance the application of a blister over the painful spot will often be all that is required locally, and a simple alkaline mixture with iodide of potassium will suffice for general treatment, *e.g.*—

R Potassii iodidi, ℥j.
 Potassii acetatis, ℥iij.
 Potassii bicarbonatis, ℥ii.
 Aquam Chloroformi ad ℥vj. M.

A tablespoonful in water three times a day.

In the second stage one is confronted with the treatment of pleural effusion. The first point to be settled is as to the advisability of paracentesis. It is not advisable to tap during the period of fever, if it can be avoided, the reason for this being that if the patient be tapped during the pyrexial stage, *i.e.* the time during which the inflammatory mischief in the pleura is still in progress, fresh fluid will almost certainly be poured out, thus necessitating another tapping. It will usually be found that by waiting ten days or a fortnight, the temperature will go down, and then the question of tapping may properly be considered. Still, there are cases in which the displacement of the heart from a very large effusion, especially if on the left side, threatens death from heart failure, and cases in which, owing to one pleural cavity being rapidly filled with fluid, or the two cavities half filled, there is congestive bronchitis of

the remaining portion of the lungs, threatening death from asphyxia. Under these circumstances paracentesis should be performed no matter how high the temperature may be. With these exceptions, and having satisfied oneself by an exploratory puncture that the fluid is not purulent, it will be prudent to try the effect of medicinal treatment before having recourse to operative procedures. If the temperature continue high, the salicylate of sodium may be continued, if, however, the temperature fall, various diuretics may be tried, as for example, iodide and acetate of potassium, or Baillie's pill (℞ Pil. hydrarg., pulv. scillæ, pulv. digitalis, āā gr. j.). It is not advisable to waste much time over the drug treatment of pleural effusion when we have so potent a means as paracentesis at our disposal, so that if there be not marked improvement in the course of ten days or a fortnight, the chest should be tapped. Care should be taken that the aspirator is perfectly clean and in good order. It is well before using it to run a twenty per cent solution of carbolic acid through it and to follow this with hot water. The spot selected for the introduction of the needle should be washed with the solution of carbolic acid. In large pleural effusions the usual place of puncture is in the sixth or seventh intercostal space, in the posterior axillary line, according as the effusion is on the right or left side; but in small effusions, or where the lung is bound down by adhesions, it may be necessary to select some other spot. It is advisable to make a preliminary exploration with an hypodermic syringe to settle definitely the existence of fluid at the spot where the paracentesis is to be made. A trocar and cannula should always be employed for aspirating the chest, as if a sharp-pointed cannula be used the point may come in contact with the lung, after a certain quantity of the fluid has been withdrawn, and by tearing it give rise to pneumothorax. If the point of the trocar be sharp and the cannula of moderate size, it is hardly necessary to incise the skin, provided the trocar be plunged in boldly, so as to pierce the usually thickened pleura, and not to push it in front of the instrument. Care should be taken to keep close to the upper edge of the rib, so as not to wound the intercostal artery. This accident has been followed by death from hæmorrhage. Various measures have been employed for the production of local anæsthesia, so as to diminish the pain of the puncture. The two most in vogue at present are ethyl chloride and eucaine. The former is supplied in capsules terminating in a fine tube, with a screw-capped point, which is directed to the required part, when the chloride is volatilised by the warmth of the hand. A five per cent solution of eucaine is

taken and sterilised by boiling. Twenty to thirty minims of the solution are injected into the skin over the point selected for puncture.

It is well not to withdraw the fluid too quickly, otherwise coughing and a sense of constriction result. If, after waiting a few seconds and resuming aspiration, these symptoms continue, or the fluid become blood-stained, then the cannula should be withdrawn and the opening sealed up with collodion on cotton wool. In cases of hydrothorax, as there is no obstacle to the expansion of the compressed lung, all the fluid may be drawn off without the distress which often accompanies aspiration in cases of pleurisy. If much fluid is being removed attention should be directed to the condition of the patient's pulse, as syncope has been known to follow the rapid removal of a large pleural effusion. In some cases a single aspiration will suffice; what little fluid remains will gradually be absorbed, and if there be any delay in clearing up of the fluid absorption may be stimulated by painting the side with tincture of iodine, or applying a blister, and giving diuretics. The question arises in exceptional cases how many times aspiration should be employed. The rule that the writer has followed has been to aspirate as long as the fluid continues serous. In a recent communication on the subject Dr. Samuel West has recorded a case of serous pleural effusion of fifteen months' standing in which, after three tapplings, he resorted to free incision, of course rendering the discharge purulent: the patient made a good recovery. In some cases what has been termed a "dry tapping" occurs, *i.e.* the fluid does not flow through the cannula. If the cannula have entered the pleural cavity and there be an effusion, fluid must flow if there be a sufficient vacuum in the aspirating bottle, unless the cannula be stopped by a plug of lymph or otherwise blocked. A more common occurrence is for the fluid to cease running, although the physical signs show that there is still a large effusion. Sometimes the removal of a small quantity of fluid will start absorption; if not, the chest may be tapped again in a week or ten days' time, and then the fluid will often run freely, the preliminary tapping having allowed the lung to expand.

Fainting not infrequently occurs during or after paracentesis, and sudden death from syncope has in very exceptional cases resulted. The occurrence of profuse albuminous expectoration should be mentioned as a possible result of thoracentesis. The probable explanation of this rare and fatal complication of thoracentesis is that, owing to some vaso-motor change in the capillary vessels of the

lung as a consequence of its rapid expansion, there is a serous exudation into the alveoli, and the patient usually dies from suffocation. As a rule the attack comes on about ten minutes after the completion of the operation, but it may be delayed for an hour or two, or follow immediately. The expectoration may continue for fifteen minutes or last for hours, and it may amount to two or three pints.

Sending the patient during convalescence to a high altitude for a few weeks will much facilitate the expansion of the lung.

In empyemata the only safe plan of treatment is to freely drain the cavity, but before incising the chest it is advisable to make certain of the existence of pus by an exploratory puncture. Having done this, an incision should be made over the dull area, and a portion of one or two ribs, about 1 to 1½ inches long, should be removed. The operator should introduce his finger into the cavity, and remove any masses of puriform lymph which can be felt. A drainage tube is left in, the side well covered with an antiseptic dressing, and lightly bandaged. Whenever fluid comes through, the dressing should be reapplied. As a rule, the temperature comes down soon after the operation, and in the course of two or three weeks, if the drainage be good, the tube may be withdrawn, and recovery takes place. If the tube be left in longer than absolutely necessary, the case is much protracted. If the discharge have an unpleasant odour, this indicates imperfect drainage, possibly due to retention of pus by adhesions. The patient should be again anæsthetised, the opening enlarged, and the pleural cavity carefully explored by the finger. Washing out the cavity with antiseptic solutions has been given up on account of the danger of sudden death, which has been known to follow this procedure. A child of five who had been operated on for empyema, and whose temperature did not come down as is usually the case, was placed in a bath at a temperature of about 100° F., water being taken out and added until the water in the bath remained nearly clear. After a bath on two or three successive days the temperature fell to normal, the empyema ceased to discharge, and the opening rapidly closed up. Should an empyema continue to discharge, the cavity may be syringed out with astringents, but since free drainage has been introduced this plan of treatment is hardly ever necessary. In old-standing cases, in which the lung is bound down by adhesions, and shows no disposition to expand, it may be necessary to remove portions of several ribs in order to allow the chest-wall to fall in. In cases of double empyema the side which

contains most pus should be incised first, and the other side should be incised a week or ten days later.

Drugs are of little or no use in cases of empyema, but quinine or other tonics may be given if the appetite flags; convalescence is accelerated by sending the patient to the sea-side or other bracing place as soon as possible.

PNEUMOTHORAX—PYO-PNEUMOTHORAX

By PNEUMOTHORAX is understood the presence of air in the pleural cavity.

PYO-PNEUMOTHORAX.—This signifies the existence of air and pus in the pleural cavity. Under this heading it will be convenient to consider cases in which fluid and air co-exist in the pleural cavity, whatever be the nature of the fluid.

Clinically two varieties of pneumothorax require consideration:—

1. Pneumothorax occurring in persons apparently healthy.
2. Pneumothorax occurring in persons whose lungs are affected.

When pneumothorax occurs in apparently healthy persons the **symptoms** presented by the patients are usually quite characteristic. The attack comes on suddenly, generally after some exertion, the patient complains of severe pain in the chest, great shortness of breath, and a sense of suffocation. The respirations are laboured and hurried, there is usually a troublesome cough, but without expectoration. The pulse is small and frequent. The patient is pallid, or may be cyanosed, and the skin is clammy. The posture of the patient varies; in some cases the recumbent position cannot be assumed without causing symptoms of suffocation.

In those cases in which pneumothorax occurs in persons with lungs already much damaged, if the side most diseased be involved, the escape of air into the pleural cavity may take place without any marked symptoms. Under these circumstances the pneumothorax is limited by pleural adhesions. If the lungs were previously only slightly affected, the onset of pneumothorax will be attended with much the same set of symptoms as in the first group. In pyo-pneumothorax, in addition to the symptoms mentioned, the patient suffers from cough attended in some cases by the expectoration of foetid, puriform sputa. There is frequently cyanosis. The patient lies on or towards the affected side. In cases which have lasted some time there is great emaciation and other hectic symptoms.

When death takes place within the first week it is usually due

to suffocation, though sudden death from shock has been recorded. If it occur later it may be due to a complication such as empyema, or to the original malady which caused the pneumothorax.

The physical signs of a complete pneumothorax are very characteristic, but if the pneumothorax be only partial, the escape of air being limited by pleural adhesions, the signs are less marked. If positive intra-thoracic pressure be present, the intercostal spaces are obliterated, the heart is displaced, and if the effusion be on the left side there is abolition of the cardiac dullness, the diaphragm may also be displaced downwards, and the liver and spleen may consequently be felt below the costal arch, in right and left pneumothorax respectively. The percussion note is tympanitic over the affected side, though it is stated that when there is great increase of intra-thoracic pressure the percussion note has been found to be impaired; this condition the writer has never detected. Vocal fremitus is absent. On auscultation there may be entire absence of all respiratory murmur, but in some cases amphoric breathing may be heard over a limited area. Vocal resonance is usually lost, but the voice sounds may have an amphoric character, or bronchophony or pectoriloquy may be present. The bell-sound (*le bruit d'airain*) may generally be elicited. In exceptional cases metallic tinkling has been audible. Puerile breathing is usually present over the sound lung.

In pyo-pneumothorax the physical signs are the same as in pneumothorax, excepting that there is dullness on percussion over the lower part of the chest, and tympanitic resonance above the level of the fluid. Metallic tinkling and a splashing sound on succussion are also frequently heard. The latter is a conclusive evidence of the presence of fluid and air. Metallic tinkling, on the other hand, has occurred in cases in which there was no other evidence of fluid in the pleural cavity.

Diagnosis.—Pneumothorax may be confounded with emphysema, this could only be the case if the emphysema were much advanced; now advanced emphysema is invariably bilateral, but bilateral pneumothorax is incompatible with life, hence there should be no difficulty in excluding emphysema. A large, superficial cavity may be confounded with a limited pneumothorax, in fact all the signs met with in the latter may occur in the former, and as pneumothorax is most commonly due to pulmonary tuberculosis the history of the case may give no assistance. In cases of excavation, however, there will almost certainly be retraction of the chest-wall, whereas in pneumothorax there is usually some fulness over the

affected part of the chest. The position of the cardiac impulse is of great importance; with a cavity the apex is drawn to the affected side, whereas in pneumothorax it will probably be displaced to the sound side. The entry of the stomach and intestine into the pleural cavity, through a rent in the diaphragm, has simulated pneumothorax; a definite diagnosis in a case of this kind is practically impossible. Dulness below, resonance above, and splash on succussion, present a combination of signs absolutely diagnostic of pyo-pneumothorax.

Prognosis.—In cases of pneumothorax occurring in the apparently healthy, the immediate prognosis is distinctly good, as nearly all the cases recover in from five days to six weeks, but it must be remembered that many of these will probably develop phthisis later on. The mortality of pneumothorax from all causes is, however, very high, less than 30 per cent of the cases recovering; the mortality is greater during the early hours and days of the disease, so that, other things being equal, the chance of life increases in proportion to the time which has elapsed from the onset. The cause of the pneumothorax and the condition of the opposite lung should exercise a powerful influence on the prognosis. Inasmuch as phthisis is the usual cause, the mortality in these cases will be the average one. Should the other lung be much diseased, it will be unable to respond to the extra call upon it, and therefore the patient will have but a poor chance of life. In gangrene of the lung the prognosis is practically hopeless.

The presence of pus in the pleural cavity adds very much to the gravity of the prognosis, it indicates a more serious condition of affairs and there will be in addition the special dangers attendant on the surgical treatment required in these cases.

Treatment.—In cases of pneumothorax, in which there are symptoms of shock, the administration of diffusible stimulants is indicated. The most speedy and effective procedure is the subcutaneous injection of twenty to sixty minims of ether, or a subcutaneous injection of the $\frac{1}{50}$ to $\frac{1}{30}$ of a grain of the hydrochloride of strychnine. Cases accompanied by severe pain require the subcutaneous use of morphine, $\frac{1}{6}$ to $\frac{1}{4}$ grain. In order to unload the portal system and at the same time to prevent the patient straining at stool, aperients calculated to produce a loose evacuation should be given.

Paracentesis is required when there is excessive dyspnoea or failing action of the displaced heart, but it is best avoided if possible. A single paracentesis usually suffices to give relief, but

the operation may have to be repeated. Paracentesis is best performed with a simple trochar and cannula, to the latter is attached a tube with the end under water. In order to prevent air escaping into the cellular tissue and thus causing surgical emphysema, care should be taken to press the finger over the puncture as the cannula is withdrawn, and the opening should be sealed with antiseptic wool and flexible collodion.

Marked venous engorgement would suggest the propriety of dry cupping or venesection.

As long as there are any signs indicative of pneumothorax the patient should be kept absolutely at rest in bed.

The above remarks refer more particularly to cases of pneumothorax occurring in persons apparently healthy, but with modification they are applicable to cases of limited pneumothorax. If the lung on the affected side were known to have been much diseased this would be an additional reason for abstaining from paracentesis. Indeed, Dr. Murphy of Chicago has suggested the artificial induction of pneumothorax in cases of phthisis, by the injection of nitrogen into the pleural cavity, as a curative measure.

Pyo-pneumothorax requires to be treated on the same lines as an ordinary case of empyema, viz. by drainage. If the dyspnoea and other symptoms are urgent and are not relieved by paracentesis, incision may be necessary in the early stage of pyo-pneumothorax; in the later stage incision is always necessary if the effusion be purulent, and it is occasionally required when the effusion is serous and very chronic.

The success of the operation for pyo-pneumothorax depends upon the expansive power of the lung. Hence, in old-standing cases, where the lung is bound down by adhesions, or in cases in which the lung is riddled with cavities, operative interference should not be attempted.

HYDROTHORAX.—The symptom which attracts most attention is shortness of breath, but the effusion often takes place so gradually, and is so over-shadowed by the symptoms of the original malady that, unless detected by careful physical examination, it is frequently only discovered at the necropsy. There is no pleural friction and consequently no pain in the chest. The only points in which the physical signs differ from those of an ordinary pleural effusion are that the effusion in hydrothorax is usually bilateral, and that position has more influence on the area of dulness, owing to the absence of pleural adhesions.

If there be much fluid and the breathing be embarrassed, the

chest should be aspirated, although it is almost certain that the fluid will recur.

HÆMOTHORAX.—In cases of sudden extravasation of blood into the pleural cavity in large quantity, there will be pallor and syncope, otherwise the only symptom which attracts attention is shortness of breath.

The physical signs are those of a pleural effusion.

It is not desirable to interfere surgically with hæmothorax.

CHYLOTHORAX.—The symptoms and physical signs are, for the most part, those of pleural effusion. The diagnosis can only be made by an exploratory puncture. Removal of the fluid by paracentesis is the only treatment to be pursued.

TUBERCULOSIS OF THE PLEURA

According to Hodenpyl,¹ miliary tuberculosis of the pleura is of frequent occurrence; thus he states that out of ninety-one cases, in which the lungs were free from tuberculosis, in forty-five, or nearly 50 per cent, there were seen on the surface of the pulmonary pleura certain nodules which previous studies had led him to regard as being tuberculous in character. The gross diagnosis was confirmed by microscopic examination in all but four of these forty-five cases. If tubercle bacilli escape into the pleural cavity, an acute exudative inflammation, with the production of a sero-fibrinous or purulent fluid, may result. This condition is termed acute tuberculous pleurisy. In some cases the disease sets in suddenly with rigors, high temperature, and pains in the side, and if occurring in previously healthy persons the tuberculous origin of the attack may not be suspected. Secondary tuberculous pleurisy, in which extension takes place from pulmonary tuberculosis, is very common.

MORBID GROWTHS IN THE PLEURAL CAVITY

As already mentioned malignant disease may occur either primarily or secondarily in the pleural cavity. The former is comparatively rare, but the writer has recorded a case in which the whole of the pleural cavity was occupied by a round-celled sarcoma, the lung being completely collapsed. The physical signs in this case were entirely those of an empyema. Where there is no primary disease to attract attention, cancer of the pleura may be

¹ *Proc. New York Pathological Society*, 12th April 1899.

suspected when the fluid drawn off on exploratory puncture is blood-stained. Occasionally distinct nodules can be felt in the thoracic parietes. If there be great dyspnœa, aspiration should be resorted to, not of course with any idea of curing the patient, but simply for the relief it affords.

HYDATIDS OF THE PLEURA

In this country hydatid of the pleural cavity is a rare disease, but in certain parts of the world, notably Australia and Iceland, it is so common, that whenever there is evidence of a growth in the chest, the rule should be to exclude hydatid disease before other conditions are considered.

Inasmuch as hydatid of the pleura is almost always secondary to hydatid disease of the liver, primary hydatid of the pleura being a very rare condition, there is usually the history of the primary affection to assist in the diagnosis; indeed, it is to the history of the case that we must trust, as there are no symptoms or physical signs pathognomonic of hydatid of the pleura. The diagnosis may be clinched by the use of the exploring syringe. The most common mistake to make is to regard an hydatid tumour in this situation as an example of pleuritic effusion rather than the converse. The insidious growth of an hydatid, and the absence of constitutional symptoms which attend its progress, should be remembered. The co-existence of pleuritic effusion with hydatid tumour is a possibility to be borne in mind.

As there is a special risk of rapid suffocation in treating these cases by aspiration, it is advisable to cut down, to excise a portion of one or more ribs, and to open and drain the cavity.

F. DE HAVILLAND HALL.

DISEASES OF THE MEDIASTINUM

MEDIASTITIS

Inflammation of the cellular tissue of the mediastinum may be acute or chronic.

ACUTE MEDIASTITIS is a rare condition, and very little is known as regards the causation of the disease. In some cases it is to be attributed to pneumococcal infection; but other organisms may be responsible. In a case seen by the writer, the patient, a woman of forty, was seized with acute pain down the sternum, rapid rise of temperature, and frequent pulse. In a few hours there came on great shortness of breath and pain in the side on breathing: on examination, pericardial and pleural friction were recognised. Swallowing was intensely painful. The mind remained quite clear until death, which occurred fifty-four hours from the onset. The only explanation of the attack was that the patient had swallowed a bone two days previously, but no signs of injury to the pharynx or œsophagus were found at the necropsy. The cellular tissue of the mediastinum was infiltrated with puriform lymph, and pericarditis and pleurisy were present. The whole process was of an acute septic nature. In a typical case like the above, a diagnosis can be made; usually, however, there is extreme difficulty in making a definite diagnosis. The presence of "mediastinal crepitation," which can sometimes be elicited on deep inspiration, but which is at other times of cardiac rhythm, would be of diagnostic value.

If the affection does not prove fatal the symptoms may subside, and chronic mediastinitis may be the result, or suppuration may occur, giving rise to an abscess in the mediastinum. In virulent forms of infection gangrenous mediastinitis is met with. Abscess of the mediastinum is of much more frequent occurrence in the anterior than the posterior mediastinum. The most constant symptom of mediastinal abscess is the deep-seated pain referred to the post-sternal region, and there is usually some superficial tenderness. The pain increases in severity, seldom abating until the pus has found an outlet. The pulsating character of the pain is often well marked: this is probably due in great measure to pulsation transmitted from the heart or great vessels. The usual constitu-

tional symptoms attending the formation of pus are present. In chronic abscess pressure symptoms, such as dyspnœa and œdema, are more prominent than pain.

On physical examination nothing may be detected, but if the abscess be large, a fluctuating tumour may be felt above the episternal notch, or in one of the upper intercostal spaces, usually on the left side, and this bulging may have an impulse on coughing, or pulsation may be communicated to it from the heart. The symptoms of abscess of the posterior mediastinum are somewhat vague: there may be local pain and tenderness, and possibly dysphagia, from pressure on the œsophagus. Physical examination does not render much assistance in the diagnosis: attention should be directed to any possible cause of the complaint. The importance of not confounding abscess with aneurysm, and *vice versâ*, should be always borne in mind, as fatal mistakes have been made in the past.

The **prognosis** of mediastinal abscess is very grave, but not so bad as for cancer or sarcoma of the mediastinum. If the anterior mediastinum be the part affected, the prognosis is better than when the disease is seated elsewhere, as the pus is more superficial, and therefore, more accessible to surgical interference. One great danger is the possibility of pus making its way into the lungs or pleural cavities.

Etiology.—Suppurative mediastinitis may be set up by the extension of inflammation from the neck, as after tracheotomy, or as a result of “angina Ludovici.” Tuberculous disease of the lymphatic glands is, however, the most common cause. It may follow the exanthemata, especially enteric fever and measles, or be the result of extension from septic forms of pneumonia and pleurisy. Many cases own a traumatic origin, either from blows on the sternum, or from the impaction of foreign bodies.

Treatment.—In cases of acute mediastinitis the treatment applicable to inflammation of other parts of the body must be carried out. Rest in the recumbent position must be enforced; if pain and tenderness are complained of, poultices, hot fomentations, or glycerine and belladonna, may be applied. Opium internally, or morphine subcutaneously, may be required for the relief of pain and insomnia. If the temperature be high, an occasional dose of antipyrin or phenacetin will probably relieve the patient. Quinine and other tonics may be necessary. Should an abscess form, surgical interference is generally called for, as those cases of abscess which have been operated on, and freely drained, have recovered, whereas if pus remain pent up in the mediastinum,

a fatal termination of the case is almost inevitable. Several cases of successful trephining of the sternum have been recorded.

CHRONIC MEDIASTINITIS (INDURATIVE MEDIASTINO - PERICARDITIS).—This, though a comparatively rare affection, is more often met with than the acute variety.

The **symptoms** are of a very varied character: shortness of breath, and a sense of constriction and vague pain in the chest, are usually present; and if they occur in conjunction with duskiness and cyanosis of the face, puffiness about the eyelids and lips, œdema of the thoracic wall, and prominence of the veins of the neck and arms, they are suggestive of this disease. The pulse is invariably increased in frequency, and the pulsus paradoxus is not uncommonly to be recognised, though it has not the diagnostic importance which was at one time attributed to it. Cough of an explosive character has been met with, and may be very exhausting. Ascites is a very common complication, and œdema of the arms and legs has been observed in some cases. Albuminuria is not a constant symptom, but is often present.

The physical signs are neither marked nor constant. There may be impairment of the respiratory movement and dulness over the affected area. Creaking, audible on auscultation when the patient moves the arm, is said to be of diagnostic value. Inspiratory swelling of the veins of the neck is of great significance. Bronchitic sounds may be heard. The signs indicative of adherent pericardium or of pleurisy are frequently present.

Etiology.—Though there is reason to believe that in most cases the commencement of the disease dates from childhood, its effects are not commonly recognised until early adult life. Simple chronic mediastinitis, in which the morbid changes are confined to the mediastinum, the pericardium not being involved, occurs later in life. Males are more frequently affected than females, in the proportion of four to one. There appears to be no particular disease which can be looked upon as especially the cause of chronic mediastinitis. In some cases the attack can be traced to an acute chest affection accompanied by pericarditis; acute rheumatism is consequently one of the antecedent conditions. It occasionally follows upon injuries. The frequent concurrence of chronic peritonitis with chronic mediastinitis is worthy of notice.

Treatment.—As the malady when fully developed is not susceptible of medical or surgical treatment, it behoves the practitioner to prevent the onset of so insidious a disease, if possible. Hence in acute rheumatism and affections of the pericardium and

pleuræ, especially in childhood, the patient should be kept in the recumbent position during convalescence for a longer time than was formerly considered necessary.

DISEASES OF THE MEDIASTINAL LYMPHATIC GLANDS

SIMPLE LYMPHADENITIS.—In connection with inflammatory affections of the neighbouring organs, the lymphatic glands of the mediastinum may become swollen and inflamed. The persistence of the paroxysmal cough in some cases of whooping-cough has been attributed to irritation of the vagus by enlarged glands.

SUPPURATIVE LYMPHADENITIS.—In cases in which the inflammatory process is of an infective nature the lymphatic glands may suppurate, and the abscess thereby formed may calcify or burst into the œsophagus, bronchus, or even the aorta.

TUBERCULOSIS OF THE MEDIASTINAL LYMPHATIC GLANDS.—These glands are very liable to become tuberculous by infection from the lung or pleura, and more rarely from bone.

If the glands become much enlarged, they may be recognised by giving rise to a dull note over the sternum. Dr. Eustace Smith has pointed out that in children a venous hum may be audible at the root of the neck when the head is thrown back, and that this is due to pressure by enlarged glands on the venous trunks. The occurrence of noisy or stridulous breathing and attacks of "croupy" cough, or paroxysms resembling those met with in whooping-cough, should suggest the possibility of enlarged bronchial glands.

NEW GROWTHS OF THE MEDIASTINUM

Etiology.—Nothing is definitely known as to the causation of mediastinal tumours. They occur at all periods of life; they have even been met with very exceptionally in early childhood. Sarcomata are more frequently seen in young people, and carcinomata in later life. The majority of cases of mediastinal tumours have occurred under forty. They are perhaps rather more common in females, whereas aneurysms of the aorta are much more common in males.

At one time the majority of the tumours met with in the mediastinum were considered to be carcinomatous, but recent investigation has demonstrated that most of the growths belong

to the class of sarcoma or lymphoma, or partake of the structure of both, and are therefore called lympho-sarcoma. The lymphomata occur as masses of enlarged glands, as in Hodgkin's disease or in tuberculosis. Gummatous tumours are occasionally met with.

Symptoms.—These vary very much according to the size and position of the tumour. As a rule the larger the growth the more pronounced the symptoms, but occasionally a small nodule may, from its position, simulate a much larger growth. The symptoms of mediastinal tumours are almost entirely the result of pressure, either direct or indirect. Pain is one of the most common of the symptoms, still it is not essential, as numerous cases have been collected in which it has been absent throughout the whole course of the disease. It may be so slight as only to be elicited on inquiry, or be extremely severe. The pain may be limited to one side or radiate over the chest and down the arms, it may be either dull, or lacinating in character; in some cases the pain comes on paroxysmally, resembling an attack of angina pectoris. Unilateral sweating and herpetic eruptions have been observed in connection with severe pain following the course of the intercostal nerves. Dyspnœa is usually an early and constant symptom, it tends to increase in severity with the growth of the tumour, and it is often paroxysmal with bouts of distressing orthopnœa. The dyspnœa may be due to pressure on the trachea, or on one or other primitive bronchus; or pressure on both pneumogastric or recurrent laryngeal nerves may cause laryngeal stenosis from paralysis of the abductors of the cords. Changes brought about in the lungs, as the twofold result of the pressure and irritation of the new growth, and obstruction to the vascular system by pressure on the blood-vessels, may also cause dyspnœa. In cases in which there is pressure on the trachea or paralysis of the abductors, the breathing is of a stridulous nature. Obstruction in the trachea is characterised by an absence of respiratory excursions of the larynx. Hoarseness may be present, due either to pressure upon the pneumogastric or recurrent laryngeal nerve, causing the corresponding vocal cord to assume the cadaveric position, or to chronic laryngitis resulting from the extension upward of inflammatory changes in the trachea or lungs. Cough is almost invariably present at some period of the disease. Occasionally it is of a harsh, clanging nature, causing the patient much distress; in other cases it is paroxysmal, and resembles whooping-cough. In cases accompanied by paralysis of one vocal cord the cough may be

hoarse or even aphonic. The character and amount of expectoration vary very much. Some patients are tormented by a constant, irritating cough without any expectoration, or with only a few pellets of viscid secretion; in other cases there may be copious muco-purulent expectoration. Profuse hæmoptysis is not a common symptom, though the sputa may not infrequently be tinged with blood. Prune-juice sputa, when present, are very suggestive of malignant disease. In exceptional cases the hæmorrhage may be so profuse as to cause death from suffocation or from mere loss of blood. A gangrenous condition of the lung will be revealed by the fœtor of the breath and expectoration. Palpitation and discomfort in the cardiac region, in some cases amounting to angina, and syncopal attacks, are complained of. In a few instances dysphagia has been a prominent symptom; this is especially the case when the posterior mediastinum is affected. Emaciation, which is a fairly constant symptom of mediastinal tumours, is, of course, very marked when there is any obstruction to the lumen of the œsophagus. An elevation of temperature is met with in some cases; this, as a rule, is more marked in cases of lymphadenoma than in cancerous disease. The rise of temperature is probably frequently due to intercurrent attacks of pleurisy. In making a physical examination it is most important to note the combination and succession of the physical signs. On inspection marked distension of the superficial veins may be seen; usually the neck and upper extremities are chiefly affected, but, as in a case in which a lympho-sarcomatous mass extended along the heart and exerted pressure on the inferior vena cava, œdema of the lower extremities may be the most prominent symptom. Clubbing of the fingers is sometimes seen. On examining the chest, loss of movement, diminished or increased bulk of one side, obliteration or retraction of the intercostal spaces, and the presence of local bulging may be noticed. Contraction of the affected side is met with, especially in cases of pressure on the main bronchus. The apex beat may be much displaced. The results obtained on palpation vary; if the growth completely obliterate the main bronchus, there is entire absence of vocal fremitus, but where the lung tissue is infiltrated and solid without the bronchi being compressed, vocal fremitus is increased. Occasionally a pulsation, communicated to the tumour by the heart or aorta, may be distinguished. On percussion there is a marked feeling of tactile resistance if the tumour be in contact with the chest-wall. In some cases an amphoric or cracked-pot note is elicited, probably due to healthy lung being compressed by the

tumour. Dulness may be the result of the bronchus being obliterated by pressure. The existence of emphysema, pleural effusion, or œdema of the chest-wall much interferes with the results obtainable by percussion. On auscultation the breath sounds will be found to be feeble or suppressed over a mediastinal growth. In cases in which the trachea is compressed the breathing will be stridulous. Pressure upon the bronchi without obliterating them may give rise to bronchial breathing. Vocal resonance, like vocal fremitus, is usually absent. The sounds of the heart are sometimes so loudly transmitted by a solid growth as to suggest the presence of an aneurysm. Feebleness or even extinction of the radial pulse on one side may be due to pressure on the corresponding subclavian artery. Inequality of the pupils may also be at times noticed, due, as in the case of aneurysm, to compression of the cervical sympathetic. On making a laryngoscopic examination, paralysis of one or both vocal cords may be seen. In some cases it is possible to distinguish narrowing of the trachea, and perforation of the trachea by a growth has been recognised during life.

Diagnosis.—Mediastinal tumours have to be distinguished from aneurysm, pleural and pericardial effusion, and from mediastinal abscess. Symptoms will give but little aid in distinguishing between mediastinal tumours and aneurysm. As a rule the onward progress of malignant tumours of the mediastinum is more sure and steady than is the case in aneurysms, but there are exceptions to this rule. The youthful age of the patient, female sex, a healthy state of the arteries, and an absence of a history of over-strain and syphilis would militate against the diagnosis of aneurysm. In favour of a tumour would be the absence of the sounds of the heart or of any impulse over the dull area, especially if this be large, and the evidence of the extension of the tumour in several directions at the same time. The existence of glandular swellings or of nodules on the thoracic wall would also be in favour of a tumour. For the diagnosis of an aneurysm the diastolic shock, especially when combined with pulsation, is of the greatest value. Tracheal tugging will also help. Severe pain, particularly in the back, is in favour of aneurysm.

The limitation of dulness to the apical region and the comparative clearness of the base, irregularity in the dulness, resonant patches occurring in the midst of the dull region, the presence of unilateral œdema, swellings or nodules in the chest-wall, point to mediastinal tumour as against pleural effusion. The withdrawal of

a blood-stained fluid with the aspirator is in favour of pleurisy set up by a malignant growth.

A pericardial effusion can usually be readily excluded. The position and uniform outline of the dulness, together with the displacement of the apex beat met with in pericardial effusion, present a combination of signs quite unlike anything seen in mediastinal tumours.

From the above it will be gathered that the diagnosis of a mediastinal tumour is oftentimes a matter of great difficulty; to decide as to the nature of the growth is usually impossible, unless we have some history of a past condition which would give us the clue, as there is no special symptom or sign indicative of cancer or sarcoma as opposed to other tumours of the mediastinum.

Prognosis.—In the case of malignant growths the tendency is slowly but surely in a downward direction, and death usually occurs in about three to six months after the commencement of symptoms of pressure, and life is seldom prolonged for over a year. Owing to the impossibility of diagnosing the nature of the growth it is advisable to give a guarded prognosis, for should the symptoms be due to a gumma they may clear up under the administration of iodide of potassium, and enlarged bronchial glands occasionally subside.

Treatment.—This must be of a palliative nature, for there are no means of effecting a cure except in those rare cases in which the tumour is of a gummatous nature, and in these cases iodide of potassium in full doses will generally have a marked effect. Even in the non-syphilitic cases iodide of potassium will often benefit the patient and should therefore be tried. Paroxysmal attacks of dyspnœa may be relieved by a whiff of chloroform. For the relief of pain, morphine in combination with atropine, injected hypodermically, is the most effective means. The inhalation of amyl nitrite or the internal use of nitro-glycerine will be found of service in anginal pains. Rubbing in a mixture of equal parts of the liniments of aconite, belladonna, and chloroform, or the liniment of menthol, will sometimes relieve the radiating pains over the chest. The question of tracheotomy has occasionally to be considered: if the dyspnœa be due to spasm of the adductors, or paralysis of the abductors of the cords the operation would be of service, but as a rule there is direct pressure upon the trachea, and the operation is consequently of no use in these cases. Should there be dysphagia from pressure on the œsophagus, the patient's nutrition must be maintained by rectal feeding, or gastrotomy may be

advised. Pleural or pericardial effusion may be treated by aspiration, if there be urgent dyspnœa. Even with the heroic surgery of the present day there is but small opportunity for operative procedures, though if the growth start from the sternum it may be possible to remove it.

F. DE HAVILLAND HALL.

DISORDERS OF THE DIAPHRAGM

The diaphragm, which forms a septum between the thoracic and abdominal cavities, consists of a central tendinous portion and a circumferential ring of muscular fibres, which, passing outwards from the tendon, are attached to the posterior surface of the ensiform cartilage, and to the cartilages of the six lower ribs and sometimes to the ribs themselves, posteriorly to fibrous tissue ligaments which arch over the psoas and quadratus lumborum muscles, and to the upper lumbar vertebræ by two thick musculo-tendinous pillars or crura. It is pierced for the passage of the gullet, the aorta, inferior vena cava, azygos vein, the splanchnic and trunk of the sympathetic nerves and the thoracic duct, and is covered above by the pleural and pericardial membranes and inferiorly by the peritoneum. Forming an unequally arched roof to the abdomen, the right vault reaches somewhat higher than the left (see Table, facing p. 1). In expiration the muscular portion, which is directed upwards with a varying slope from the bony attachments to the central tendon, lies almost in contact with the ribs, separated only by the attached layers of the parietal pleura which are in apposition, but on contraction the muscle flattens and separates away from the parietes, the intervals being occupied by the expanding lungs. Indications of this action of the diaphragm and of the lungs may be visible over the lower part of the thorax, distinct from the movement caused by the descent of the liver and other viscera. In quiet breathing the tendon descends but little, but on forced inspiration it is drawn down to an appreciable extent. In the adult the ribs are prevented from being drawn inwards when the diaphragm contracts, partly by their rigidity and still more by being fixed by the inspiratory muscles and those attached to the lower ribs; but in children, where the ribs are less resistant, and especially in rickets, the ribs are drawn inwards along the line of attachment of the diaphragm, thus forming a more or less well-marked groove (see Vol. II. p. 255, Harrison's sulcus).

The nerve supply of the diaphragm is almost entirely derived from the right and left phrenic nerves, which are distributed to the corresponding halves of the muscle, the lowest intercostals contributing a few twigs. The phrenic nerve is mainly formed from the fourth cervical root, with additional fibres from the third or fifth, or from both.

The Diaphragm is very rarely the seat of any distinct morbid change. Inflammation of the closely attached serous membranes, pleura or peritoneum, may involve to some extent the muscular fibres, causing granular degeneration and cloudy swelling with

impairment of contractility, or the fibres may undergo fatty degeneration or infiltration in common with other muscles; and some degree of atrophy may result from lesions of the phrenic nerves, or of their spinal cells of origin, or as a part of a general muscular atrophy.

Very exceptionally small cysts have been found on the surface of the muscle, probably derived from dilated lymphatic vessels. Occasionally the diaphragm may be involved in new growths which have extended from adjacent parts. But these conditions of the organ are not to be recognised during life, and only seldom suspected. The organ may be perforated by an hepatic or other sub-diaphragmatic abscess, or by a gastric ulcer, or in the reverse direction by an empyema or a pulmonary abscess. Rupture from violent straining has been recorded, and congenital deficiencies are occasionally met with. It is presumed that the diaphragm may be the seat of myalgia, but it is extremely difficult to differentiate this from a similar affection of the intercostal muscles, or even from a diaphragmatic pleurisy, the special features of which are referred to on p. 258. The disorders of the diaphragm are either in the direction of excessive (spasm) or of diminished (paralysis) motility.

SPASM OF THE DIAPHRAGM.—This may be of either the clonic or tonic variety.

Clonic spasm, commonly known as “hiccough” or “singultus,” is of much the more frequent occurrence, and consists in recurring brief and forcible contractions of the diaphragm, causing a violent inrush of air to the lungs, and this meeting the partially closed glottis is thrown into vibration, producing the well-known clicking sound; movements of the nares may also accompany the spasm. The phenomenon probably depends on a want of co-ordination between the associated movements of the diaphragm and glottis during inspiration. Normally the latter widens as the muscle descends, and should these actions correspond no noise should be produced, but when the diaphragm forcibly contracts and the abduction of the vocal cords is inconsiderable, the conditions exist for the production of a sound in the manner described. The rate of recurrence is very variable, from three or four times in a minute to fifty or more, in such cases seriously embarrassing the breathing. Considerable aching pain along the line of attachment of the diaphragm follows a repetition of the spasm, and if it be long continued will lead to grave exhaustion, seriously complicating the condition which has given rise to it. Cases of hiccough lasting for weeks, months, and even years have been recorded. When very prolonged the

patients are likely to be of neurotic temperament, and the disorder sometimes has appeared to be associated with epilepsy, replacing in some measure the convulsive fits.

The conditions which give rise to this symptom may be affections of the central nervous system, such as mental or emotional states, hysteria; toxic conditions, uræmia, the poisons of the acute infections, especially typhoid fever, or other perverted blood states due to cardiac failure; structural diseases of the central organs, meningitis or hydrocephalus. Sometimes hiccough is referable to irritation of the phrenic nerve in its course. The commonest causes, however, are to be found in reflex excitation *via* the vagus. Thus it is that over-distension of the stomach, whether from food or flatus, so frequently determines hiccough, and less often diseases of the stomach, such as carcinoma. Peritonitis, appendicitis, inflammation of the pleura, pericardium, or lungs may each one give rise to the symptom; and occasionally hepatic disease, disorders of menstruation or uterine disease underlie the phenomenon.

The prognostic significance of hiccough will obviously vary with the cause. Although very distressing in the purely neurotic cases, it is of little moment and may be continued for months with but slight interference with the general nutrition. In peritonitis and other serous membrane inflammations it is of graver import, and is especially so in typhoid fever, where it may be largely responsible for a fatal termination.

The **treatment** of this troublesome symptom is unsatisfactory, in the sense that no certainty can be placed upon any one of the many remedies that have been suggested, and it may become necessary to try them one after another with but little scientific principle as a basis.

The temporary and acute cases referable to gastric distension may be relieved by an emetic when it has failed to yield to holding the breath, sipping a glass of water, or other such means as may tend to establish a more uniform action of the diaphragm. Even in the more severe and prolonged cases, a subcutaneous injection of apomorphine (gr. $\frac{1}{25}$ to $\frac{1}{6}$) has been known to cure by the vomiting induced; a fit of sneezing or coughing, artificially induced by snuff, will act in the same way, the violent contractions of the diaphragm induced thereby upsetting and arresting those determined by central states.

Another group of remedies is to be found in the so-called antispasmodics, such as hyoscyamin (gr. $\frac{1}{120}$) subcutaneously, conium (gr. 2 of the extract repeated every six hours), tincture of opium

(5 drops every two hours for six doses, and then a wait of three or four hours, after which the doses may be repeated), or most effective of all a subcutaneous injection of acetate of morphia, or a chloroform inhalation of 20 to 40 drops.

Hot and cold applications to the spine have been tried with apparent benefit—blisters over the phrenic nerve at the root of the neck, or pressure in the same situation; hot fomentations to the epigastrium. Galvanism with the positive pole at the neck and the negative electrode over the phrenic nerve may be tried, and success has been claimed for traction on the tongue.

Tonic spasm of the diaphragm is of rarer occurrence. It may occur as the result of strychnine poisoning, or as a symptom of tetanus or of hydrophobia, and excessive laughing has been said to cause it. Probably it is most often met with in some forms of asthma, and it has been recorded as a manifestation of hysteria.

The condition is recognised by the fulness and comparative immobility of the lower part of the chest, the exaggerated movements of the upper part, and protrusion of the epigastrium and descent of the liver. There is extreme dyspnœa and pain around the thorax, corresponding to the line of attachment of the diaphragm.

Unless it be capable of relief by inhalations of chloroform or morphine subcutaneously, the symptom is apt to be speedily fatal.

PARALYSIS OF THE DIAPHRAGM is brought about by disease or injury of the spinal cord in the mid-cervical region, or by inflammation—post-diphtheritic, post-influenzal, alcoholic, plumbic—of the phrenic nerve, sometimes by pressure in its course from tumours, etc. When due to neuritis it is seldom if ever the only muscle affected. Hysteria is an occasional cause, and atrophy or degeneration of the muscle is still more rare. Full reference to this condition, which may be unilateral or bilateral, has been already made (see Vol. III. p. 251).

The most characteristic symptom of this condition is a reversal of the normal respiratory movements at the epigastrium, which sinks in inspiration and bulges outwards in expiration. Great care, however, must be exercised in coming to a conclusion on this point, for repeated observation has shown that this abnormality may take place in deep breathing without any diaphragmatic paralysis, and the state can only be positively affirmed when inspiratory recession takes place both in quiet and forced inspiration, and sneezing, coughing, vomiting, and defæcation are seriously interfered with. In deep breathing the diaphragm normally plays but a small part, the upper part of the thorax being chiefly concerned,

and hence the examination of quiet respiration becomes necessary for the measure of epigastric movement, for which palpation as well as inspection should be employed. It is possible also for a healthy person by practice to cause epigastric recession by forcibly contracting the abdominal muscles during inspiration. Hence this sign as an evidence of paralysis of the diaphragm must be used with caution. The movements of the lower ribs are usually much increased, though the total capacity of the chest is diminished by the excessive arching of the diaphragm. As a rule dyspnœa is but very slight except on exertion.

If the state be suddenly induced, and especially if there be any impairment of the movement of the intercostal muscles, a fatal result may quickly follow, and in any case its occurrence is to be viewed with apprehension. When due to peripheral neuritis of the phrenic nerve or nerves, recovery may be looked for if this be post-diphtheritic or post-influenzal, and improvement is sometimes rapid, but when of alcoholic origin the prognosis is much less hopeful. Additional danger is contributed by the liability of the bases of the lungs to become congested or collapsed from insufficient expansion and recoil, owing to the motionless diaphragm.

Only such cases as are due to neuritis are amenable directly to treatment, which is most effectively exhibited by hypodermic injections of strychnine (gr. $\frac{1}{100}$) or by electricity. Artificial respiration repeated for a short time every few hours and brandy or ether have saved life in diphtheritic cases, and for the same purpose inhalations of oxygen should be tried.

The movements of the diaphragm may be considerably interfered with by morbid conditions within the chest or in the abdomen, or occasionally in both regions. Extensive effusions into the pleural, pericardial or peritoneal cavities, large intra-thoracic or abdominal growths, adhesions of the pleural surfaces or of the liver to the diaphragm, extensive retraction of the lungs, and tight-lacing are those of most frequent occurrence.

The extent of symptoms will in some measure depend upon the degree to which the obstructing cause has attained, but still more upon the rate at which it has been established, those of slower production allowing time for the respiratory mechanism to make an attempt at adjustment. Hence the amount of dyspnœa is variable, but a tightness of the chest and sense of constriction and weight, and a difficulty in taking a full breath, or in coughing, are usually complained of.

W. H. ALLCHIN.

THE VASCULAR SYSTEM—PHYSIOLOGICAL INTRODUCTION

Summary of the first principles of the circulation—The heart valves—The pericardium—The cardiac muscle—Tone of the heart—Cardiac contraction—Arrhythmia of the heart—Innervation of the heart and pulse frequency—The cardiac impulse—Intra-cardiac pressure—Work of the heart—The sounds of the heart—The Pulse—The arterial system—Influence of gravity—Hypostasis—Capillary circulation—Relation of lymphatic to vascular system—The venous circulation—The pulmonary circulation—The cerebral circulation—The vaso-motor system.

The blood is a viscous fluid, and its viscosity varies ; it is propelled by a pump, the heart, which acts intermittently, and varies both in rate and energy ; it circulates through a system of tubes, which varies in capacity, while the walls of the tubes may alter in elasticity. Lastly, the blood continually varies, both in quantity and in quality, as it transfuses through the walls of the vascular system into the tissues, or passes back from the tissues into the blood-vessels. It is evident, therefore, that the problems of the circulation are far from simple.

Summary of the First Principles of the Circulation

1. The heart is an intermittently acting pump, and is provided with valves which direct the flow of blood from the veins to the arteries.
2. The large arteries are distensile elastic tubes, while the small arteries and arterioles are muscular and elastic tubes.
3. The capillaries are formed of a single layer of flat endothelial cells. This is of extraordinary tenuity, and through it there takes place the exchange of matter between the blood and the tissues—the exchange which depends partly on filtration and osmotic pressure, partly on physico-chemical conditions of the living cells, which still defy elucidation.
4. The veins are much more capacious than the arteries, and are provided, especially in the limbs, with valves. The veins are equally tough but less muscular and extensile than the arteries.
5. The blood flow is impeded by the “peripheral resistance.” This is due to the friction of the concentric layers of blood moving within the blood-vessels. The film of blood that wets the wall of each vessel is stationary, while in the axis of the stream the blood moves with the greatest velocity.
6. The friction in a blood-vessel is proportional to the area of surface exposure, and to the viscosity of the blood ; nearly proportional

to the square of the velocity of flow; inversely proportional to the sectional area. The friction is, therefore, greatest in the arterioles, for there the velocity of flow is high, the surface area increased, and the diameter diminished. The diameter of the arterioles, and thus the resistance, are controlled by the vaso-motor nerves.

7. Owing to the peripheral resistance the blood escapes with difficulty into the venous system. The large arteries are therefore distended by the systole of the heart, and part of the kinetic energy, with which the blood is endowed by each systole of the heart, is stored up as potential energy by the arterial wall. The elasticity of the arterial wall continues to force the blood through the arterioles during the diastolic period: in other words, the potential energy again becomes kinetic, and the intermittent flow from the heart is converted into a continuous flow through the capillaries.

8. The energy imparted to the mass of blood (systolic output) which is expelled by the heart at each systole is almost entirely spent in overcoming the frictional resistance, and is dissipated into heat. Not more than $\frac{1}{300}$ th part of the total energy of the heart is spent in maintaining the velocity of flow in the aorta. The kinetic energy of the blood varies as the square of the velocity of flow. The mean velocity of flow in the capillaries (1 mm. per sec.) is not more than $\frac{1}{300}$ of that in the aorta (300 mm. per sec.). The kinetic energy of the blood, when passing through the capillaries, is thus reduced to $\frac{1}{90000}$ of its value in the aorta. The potential energy, as measured by the lateral pressure, is reduced by at least two-thirds, viz. from 110 mm. Hg. to 30-40 mm. Hg.

9. In the veins the lateral pressure continues to fall, while, on the other hand, the kinetic energy or velocity increases. Finally, the velocity of the blood in the thoracic veins reaches a value not less than half that in the aorta. Hence the kinetic energy of the systolic output becomes in the venous course 20,000 times greater than in the capillaries. The increase of kinetic energy is due partly to the change of potential into kinetic energy, but chiefly results from other sources of energy, such as the compressive action of the skeletal and visceral muscles, the aspirating and compressive action of the respiratory pump, and the influence which gravity exerts on the circulation during every change of posture.

10. The *total* cross-sectional area of the vascular system gradually widens in passing from the pulmonary artery and aorta to the capillaries. The diameter of each capillary is not greater than $5\text{--}20\ \mu$, but the total cross-sectional area of all the systemic capillaries, full of blood at any one time, is estimated to be at least 700 as great as that of the aorta. The total cross-sectional area gradually diminishes in passing from the capillaries to the venæ cavæ and pulmonary veins, and finally approximates to that of the pulmonary artery and aorta.

11. In each unit of time the same quantity of blood must, on the average, flow through the lesser and greater circuit, for otherwise the circulation would not continue. It follows from this that the average velocity of flow, at one part of the vascular system, is inversely proportional to the total cross section at that part. Any *general* change in velocity in any part of the circuit tells both backwards and forwards on the velocity in all other parts, for the average velocity in the arteries, capillaries, and veins, these vessels being respectively taken as a whole, depends always on the relative areas of their total cross sections.

12. The pressure of the blood in the aorta is dependent on (1) the

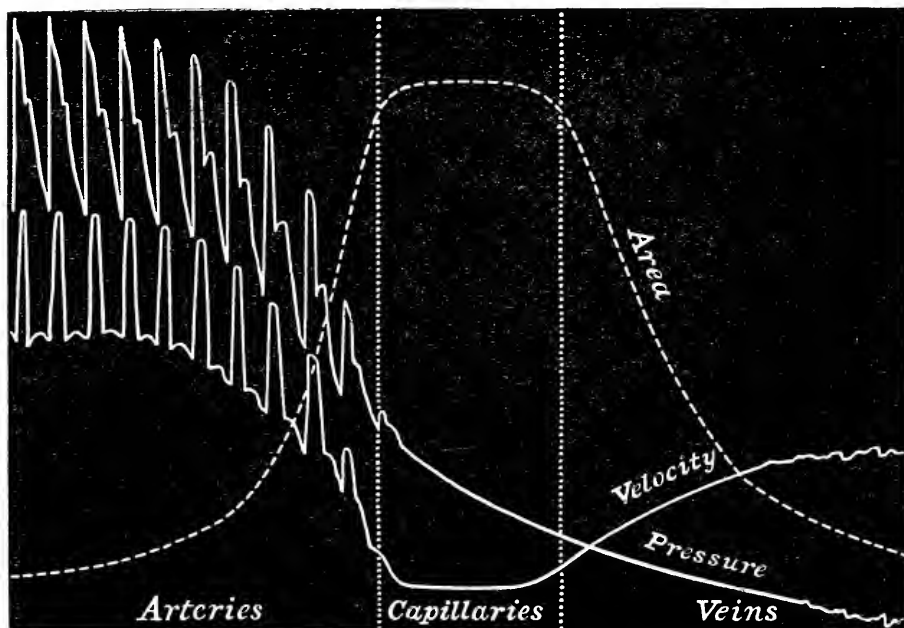


FIG. 16.—Diagram of area of vascular system and distribution of pressure and velocity during diastole and systole of the heart.—Fredericq.

energy of the heart, (2) the peripheral resistance, (3) the hydrostatic pressure due to the influence of gravity. These factors continually vary. The energy of the heart may be increased per unit of time by acceleration, or by augmentation of the cardiac contractions. The peripheral resistance varies (1) with the state of muscular tone of the arterioles, (2) with the degree of compression exerted by the action of the skeletal and more especially of the abdominal muscles, (3) with the degree of compression produced by the pressure of external objects against the body. The hydrostatic pressure due to the weight of the column of blood which occupies the vascular system, varies with the position of the body. The hydrostatic pressure of the superincumbent

column of blood adds itself in each blood-vessel to the lateral pressure produced by the energy of the heart.

13. Suppose the energy of the heart and the hydrostatic pressure remain constant, then in proportion as the peripheral resistance increases, so the lateral pressure in the aorta rises and the velocity lessens. On the other hand, as the peripheral resistance decreases, the pressure falls and the velocity increases. Suppose the peripheral resistance and the hydrostatic pressure remain constant, then as the energy of the heart increases or decreases, both the pressure and the velocity in the aorta together become greater or less.

It is obvious that, by compensatory changes taking place in the heart and the peripheral resistance, the velocity of flow in the aorta may remain constant while the pressure varies, or the pressure may remain constant while the velocity varies.

14. The vascular system is so co-ordinated by the cardiac and vaso-motor nerves that *local* dilatation in one organ is compensated for by constriction in another, while more *general* vaso-constriction or dilatation are compensated for by a diminution or increase in cardiac energy. The aortic pressure remains, except at times of muscular effort, almost constant.

15. Owing to the vaso-motor mechanism, the blood-quantum, necessary for the bodily needs, can be greatly reduced in amount, for all the available space in the vascular system need not be filled to distension. The potential space in the veins and tissue-spaces acts as a reservoir wherever there be a temporary obstruction to the blood-flow.

16. The body, taken as a whole, may be compared to a sponge full of fluid and confined by a taut and elastic skin. A varying but ever-positive pressure is maintained upon the tissue fluids by the tone of the skeletal muscles. Owing to this pressure the capacity of the venous system is adapted to the varying quantity of blood within it. Immobility of the body, loss of muscular tone, and diminished tautness of the skin alike entail increased capacity, over-filling of the venous side of the vascular system and œdema of the tissues.

17. The arteries may be compared to a high-pressure main. By means of the vaso-motor nerves the taps or arterioles can be opened or shut down, and the current switched on to or off any organ according to its functional needs. If all the arterioles be dilated at one and the same time, the aortic pressure falls, and the blood, taking the pathways of least resistance, gravitates to the most dependent parts of the vascular system.

18. If the vascular system be greatly increased in capacity by loss both of arterial tone and of the tone of the skeletal muscles, and if the body be in the erect posture, then in such case the blood, *owing to its weight*, sinks into the dependent parts. The heart may empty and the cerebral circulation altogether cease. Under such extreme conditions

the circulation of the blood continues only so long as the body is in the horizontal or inverted posture.

19. Local variations in the velocity of the circulation continually occur. For example, when the arterioles locally dilate or constrict; when the veins become more or less congested by the influence of position or the pressure of external objects; when the blood becomes more or less expressed by the contraction of the skeletal muscles.

20. Valves are set in the veins and lymphatics so that the contraction of both the visceral and skeletal muscles may further the return of the blood and lymph to the right side of the heart. Similarly, the blood which fills the expanded pulmonary vessels during inspiration is expressed from the lungs into the left side of the heart by the act of expiration. For the maintenance of an efficient circulation the activity of the whole muscular system is of not less importance than the beat of the heart, and this is especially the case when the body is in the erect posture.

21. In times of general muscular effort both the lateral pressure and velocity of flow in the aorta are raised by the increased energy of the heart. At the same time, by the greater activity of the respiratory pump and the expressive action of the skeletal muscles (which now contract and now relax), the diastolic filling of the heart is maintained. The blood is derived from the splanchnic area and driven in greater volume through the locomotor organs. This is brought about by constriction of the splanchnic vessels, by contraction of the abdominal muscles, and by the pumping action of the diaphragm.

22. General tonic spasm, as in status epilepticus or strychnine tetanus, lessens the total capacity of the vascular system, raises very considerably the pressure in every part of the vascular system, and impedes the circulation of the blood.

23. Owing to the potential capacity of the veins there occurs, when the systemic arterioles alone are generally constricted, hardly any increase in lateral pressure in either the vena cava or the pulmonary artery.

24. The venæ cavæ and portal system on the one hand, and the pulmonary system on the other hand, form low "heads of pressure," from which the right and left heart respectively are supplied. Thus the diastolic pressure is kept constant when changes in arterial pressure are brought about by vaso-constriction or increase in cardiac energy.

25. It is of the utmost importance that the heart should not be over-dilated during diastole by any increase of diastolic pressure.

When a man stands on his head the venæ cavæ and right side of the heart are liable to over-distension owing to the weight of the superimposed column of blood. The pericardium in such case limits the expansion of the heart. Over-dilatation of the right heart during a period of great muscular effort, for example, straining at stool, is prevented not

only by the pericardium but by the concomitant closure of the glottis and rise of intra-thoracic pressure. While the right heart and lung are thus supported, the blood in the abdomen is driven through the lungs into the left heart, and so into the locomotor organs, by the contraction of the powerful abdominal muscles.

26. After death the blood expelled by the post-mortem contraction of the arteries passes out of the arterial system and congests in the veins, capillaries, and tissue lymph spaces in the dependent parts (hypostatic congestion). The arteries are thus found to be empty after death.

THE HEART

The size of the normal adult heart is about 5 by $3\frac{1}{2}$ by $2\frac{1}{2}$ inches; the weight, 9-10 ozs.; the capacity of each chamber about 3 ozs. The size varies as the mass of the skeletal muscle. The right auricle forms the base of the heart in front and to the right, and its appendix slightly overlaps the root of the aorta. The right ventricle occupies the chief part of the anterior and a small part of the posterior surface, and forms the right margin of the heart. The left auricle forms the left and posterior part of the base of the heart, while the chief part of the posterior surface, the left margin and the apex of the heart are formed by the left ventricle. The heart is lined with an endothelial membrane (endocardium) continuous with the internal lining of the vascular system.

The valves.—The tricuspid valves consist of three, and the bicuspid of two triangular cusps. The bases of the cusps in either valve fuse to form an annular membrane and so become attached to the fibrous tissue which encircles the auriculo-ventricular orifices. The under surface and free edge of each cusp are attached by chordæ tendineæ to two papillary muscles. The cords attached to the free edge are the shorter. The edges of these valves, which come into apposition, are exceedingly thin and delicate, while the outer parts, which bear the full systolic pressure of the blood, are tough. The cardiac muscle, by its contraction, limits the size of the auriculo-ventricular orifices, and so maintains the competency of the valves. Incompetency may arise when the right heart is greatly dilated. The papillary muscles and chordæ tendineæ act as stays or braces, preventing over-distension of the heart. The aortic and pulmonary valves consist of three semilunar, pocket-shaped cusps. A fibrous nodule is placed centrally in the free edge of each cusp. Opposite the cusps are the sinuses of Valsalva. Eddies formed in the sinuses, during the period of systolic output, bring the semilunar valves into apposition, so that they close without noise or jar at the moment when intra-ventricular becomes less than arterial pressure. The auriculo-ventricular valves are likewise floated up by eddies, and brought into

apposition at the moment the intra-ventricular pressure surmounts that in the auricles. The valvular sounds are not produced by the closure, but by the subsequent tension, of the closed valves.

The papillary muscles come into action synchronously with the contraction of the ventricular wall. The auriculo-ventricular valves, at first ballooned outwards by the rise of intra-ventricular pressure, are pulled backwards by the action of the papillary muscles. During the systolic output, the columnæ carneæ and papillary muscles pack closely together. The extent of contraction of the wall of the heart necessary to empty the cavity is by this means lessened. The ventricles are never completely emptied, for blood remains in contact with the auriculo-ventricular valves up to the end of systole, and assures their closure. The diastolic expansion of the ventricles is due chiefly to the elastic recoil of the papillary muscles and columnæ carneæ.

The semilunar valves, during the period of output, do not swing back against the orifices of the coronary arteries, but, directed by the eddies in the sinuses of Valsalva, assume a vertical position. The heart, during the same period, shortens in all its diameters, but the apex, owing to the downward movement of the base, remains almost a fixed point. The downward movement is allowed by the longitudinal extension of the aorta and pulmonary artery.

The pericardium is a triangular bag, inextensile and of great strength. The pericardium is suspended above by the deep cervical fascia, below it is attached to the central tendon of the diaphragm. The fixation of the pericardium prevents displacement of the heart during changes of posture. The pericardium acts as the external coat of the arteries, and restrains over-dilatation of the heart. Great dilatation and hypertrophy of the heart in children frequently follow pericarditis, and the failure of the heart is found to occur when the child reaches adolescence and enters upon a life of muscular labour. In the animal world the pericardium varies in strength in proportion to the muscular development.

Cardiac muscle consists of short branching cylindrical cells cemented together so as to form interlacing bands of fibres. The external bands run obliquely from the base to the apex of the heart, and many of these bands turn inwards at the apex and run upwards either into the papillary muscles or to the base of the ventricles. These longitudinally-arranged, hook-shaped bands shorten the ventricles, while the intermediate bands are more or less ring-shaped, and run transversely round the ventricles. The muscle bands of the right ventricle envelop the left ventricle, but the latter has in addition a thick muscular coat of its own. In every piece of the cardiac wall the fibres on the inner surface cross those on the outer surface. The muscle of the ventricles is arranged so as to *wring* the blood out of the heart. The auricular muscle bands are common to both auricles. The fibrous tissue at the

base of the ventricles surrounds the auriculo-ventricular and arterial orifices, and affords the point both of insertion and origin of the auricular and ventricular muscle bands. The cardiac muscle cells possess no sarcolemma and have a centrally placed nucleus which is surrounded with granular sarcoplasm. Cross-striated fibrils, or sarcostyles, occupy the peripheral part of the cells, but these are far less differentiated than the sarcostyles of skeletal muscle. Richness in sarcoplasm denotes capacity for strenuous and continued action.

Tone of the heart.—Heart muscle, apart from its rhythmic power of contraction, possesses tone. Heat, chloroform, acids, diminish the tone, dilate the heart, and render it flaccid. Cold, alkalies (especially ammonia), strychnine, supra-renal extract, increase the tone and contract the heart.

The cardiac contraction.—Records of the action current, obtained with the capillary electrometer, show that the beat of the heart is not a tetanic spasm but a twitch rhythmically superimposed upon the tone of the heart. The twitch, as in all muscles rich in sarcoplasm, is protracted in period. The rate of conductivity of the contraction wave (5 m. per sec.) is considerably slower than in nerve (30 m. per sec.). The heart, during the systolic period, is refractory to stimulation, and so cannot be thrown into tetanic spasm by rapidly repeated excitations. Powerful tetanising currents may throw the heart into a condition of inco-ordinate fibrillar contraction or *delirium cordis*. The inco-ordinate local contractions are quite ineffective and the blood ceases to circulate. Only single shocks at the rate of 70 per minute should therefore be employed to excite the heart in states of syncope. Rhythmic compression of the chest is a far better means of resuscitating the heart. The beat of the heart is always maximal, whether it be excited by a minimal or maximal stimulus. The amplitude and energy of the contraction depend on the metabolic condition of the heart muscle. Every piece of the heart is capable of automatic rhythm. The most persistent, the least fatiguable, and most easily excitable power of rhythm is the power of the auricular muscle, especially in the neighbourhood of the venous orifices. The ventricular muscle cells are thicker, more highly striated, slower in contraction, and less responsive to excitation than those of the auricle. Hence the auricles set the rhythm of the heart. The slower excitatory process in the ventricular muscle acts, as a rule, inefficiently on the auricular muscle, and antiperistalsis can only be set up by powerful electrical stimulation of the ventricles. The excitatory wave, starting from the venous orifices, spreads over the auricular muscle and then traverses certain muscle fibres in the auriculo-ventricular septum. These are of embryonic type, and the excitatory wave is delayed in its passage at this point, so that the ventricles contract in sequence to the auricles. *Rhythmic contraction* is the automatic function of the cardiac muscle.

It commences in the embryonic heart at a time when no ganglion cells have wandered within the cardiac muscle, and continues in the ganglion-free apex of the mammalian heart when this is fed with warm defibrinated blood. It is the expression of the rhythmic oscillations which occur in the chemical stability of the muscle plasm, and thus depends on the quality of the blood circulating through the coronary vessels. The frequency of the heart is accelerated by warming the venous orifices, while only the systolic time is shortened on warming the ventricles.

Arrhythmia.—In certain conditions of altered metabolism, *e.g.* digitalis poisoning, the heart may fall into a condition of arrhythmia, and the ventricles, owing to diminished conductivity, fail to respond to every beat of the auricles. Suppose the auricles beat seven times to the ventricles' six. Every eighth auricular systole then bears the proper time relation to every seventh ventricular systole. The remaining auricular systoles occur more or less at the wrong period in the cardiac cycle. When the auricular systole occurs during the period of ventricular systole the filling of the ventricles is impeded, the output lessened, and the pulse diminished. Conversely the pulse is more ample when the auricular systole falls within the period of ventricular diastole. This condition leads to a group pulse, such as the *pulsus bigeminus* (one beat weak in three) or *pulsus trigeminus* (one beat weak in four). Arrhythmia is a sign of acute or chronic disturbance of the metabolism of the cardiac muscle.

Innervation of the heart.—The rhythm, tone, and energy of the heart are co-ordinated with the functional needs of the organs by the nervous system. The vagus centre is reflexly controlled by afferent impulses, which stream to it from the posterior root tracts, and from the cerebral cortex. It is important to note that the heart itself possesses afferent nerves, by means of which not only the frequency of the heart, but the respiration, and especially the tone of the blood-vessels, may be reflexly modified. Both the heart muscle and the bulbar centres are directly affected by changes in the pressure, temperature, and quality of the blood. The cardiac rhythm may thus be modified (1) by the direct effect of the blood on the heart muscle; (2) reflexly by the effect of the blood on the afferent nerves of the heart; (3) by the direct effect of the blood on the bulbar centres; (4) reflexly by the afferent nerves.

An increase in diastolic pressure or intravenous injection of warm saline directly stimulates the heart to increased action. The latter is a valuable means of resuscitation in syncopal conditions. A rise of arterial pressure directly stimulates the vagus centre, and lessens the frequency; conversely a fall of pressure accelerates the heart. So long as the vagus centre be in action, the arterial pressure cannot be raised by vaso-constriction much above the normal. The *depressor* fibres are the most interesting of the afferent cardiac nerves, for by their means the arterioles can be dilated and the heart relieved from over-strain.

The cardiac inhibitory fibres of the vagus arise from the nucleus ambiguus and issue by the lowest vagal rootlets. The accelerator fibres leave the cord by the second and third dorsal anterior spinal roots, and, passing to the ganglia stellata and the inferior cervical ganglia, reach the heart by superior, middle, and lower cardiac branches from the annulus of Vieussens and cervical sympathetic nerves. The vagi and acceleratores are antagonistic nerves, and both exert a tonic action. The acceleration produced by a dose of atropine (the drug paralyses the vagal nerve-endings) demonstrates the strong bridle action which the vagal centre tonically exerts over the heart. Excitation of the vagus lessens the tone and frequency, diminishes the conductivity, and increases the diastolic period and the assimilatory (anabolic) processes of the heart. Conversely stimulation of the acceleratores increases the frequency, tone, conductivity, and dissimilatory (katabolic) processes, and lessens both the period of systole and diastole. Paralysis of the acceleratores may be followed by arrhythmia, for the conductivity of the heart is thereby lessened. There is no evidence that the acceleratores can be reflexly excited. The modifications of pulse frequency, which follow the excitation of any sensory nerve, are apparently due to increase or inhibition of vagal tone. The tone of the acceleratores gains the upper hand so soon as the action of the antagonistic vagal centre is reflexly inhibited. Severe hæmorrhage, shock, intoxicants, such as ether and chloroform, decrease the stability of the cardio-vagal centre, so that acceleration rather than retardation of the pulse follows the excitation of a sensory nerve. The paroxysmal tachycardia of neurasthenics (the frequency of beat may reach 150 to 250 per minute) seems to be due to instability of this vagal centre. Visceral irritation may be the exciting cause. Since the diastolic filling becomes incomplete in the accelerated heart while the diastolic period of rest is shortened, the condition may lead to cardiac failure and syncope. There is a limit set to the possible acceleration of the pulse. Either the intravenous injection of hot saline, or excitation of the acceleratores, increases the frequency up to a certain point, but there is no summation of effect when both means are employed together. Similarly fever does not accelerate the pulse in cases of Basedow's disease. The acceleration of the heart, when provoked by a continuous and uniform means of excitation, tends to become progressively less. This tendency is often to be noted in cases of fever. Not only the febrile blood temperature, but the nature of the toxin modifies the heart rate, as is evidenced by the fact that, with an equally high temperature, the heart is more accelerated by scarlet than by typhus fever. The cardio-inhibitory centre is frequently paralysed after pneumonia or typhoid fever. The tone of the centre can be tested by a dose of atropine. An infrequent pulse occurs when the vagal centre is excited by asphyxia, acute cerebral anæmia, as in cases of cerebral compression, or an abnormal rise in

arterial pressure. It may likewise arise from stimulation of abdominal visceral nerves (*e.g.* peritonitis), from distension of the stomach, or from absorption of bile-salts. A slow pulse occurs in badly-nourished hearts—in cases where the coronary arteries are sclerosed, and in men of indolent and over-indulgent habits whose hearts have become flabby and dilated. Stimulation of the mucous membrane of the air-passages reflexly inhibits the heart, while inspiratory expansion of the lungs, sipping, swallowing, talking, increase the pulse frequency. The pulse is greatly accelerated during, and for some time after, muscular exercise, while sleep lessens the rate (especially in children). The pulse frequency is greater in small than in big men, for in the small the surface exposure, in proportion to cubic content, is greater and the loss of heat more rapid. The average rate varies with age as follows:—

Fœtus	130-140
0-1 year	130
1-2 years	115-120
3-4 „	105-110
5-9 „	95-100
9-10 „	90
16-17 „	80
Adult life	70-80

The cardiac impulse is caused by the hardening of the muscular mass of the ventricles against the wall of the thorax. During the period of rising tension the blood presses and reacts upon the ventricular wall with a force equal to that by which it is compressed. The impulse is synchronous with the beginning of systole. It is felt at a different point in each change of posture, as a different part of the heart comes in contact with the chest-wall. In the supine position the “apex beat” or lowest and outermost point of the cardiac impulse is found in the fifth intercostal space, 2 inches below and $1\frac{1}{2}$ inches to the right of the nipple ($3\frac{1}{2}$ inches to the left of the mid-sternal line). If the body be rolled on to the right side, the apex beat shifts to the right, and may occur beneath the sternum; on the other hand, it becomes evident to the left of the nipple line, when the patient is turned over on to the left side. The *form* of the impulse curve (recorded most suitably by Edgren’s cardiograph) represents neither a true volume nor pressure curve. A diffuse or large impulse does not necessarily denote a vigorous heart. It may arise from increased area of contact. The impulse may be visible in the epigastric region when the right heart is dilated. The chest-wall is sucked inwards during systole at all points, except where the heart is in contact with the parietes. The typical impulse curve can only be obtained from the latter point.

Intra-cardiac pressure.—Taking 75 as the average number of heart-beats per minute, each *cardiac cycle* will occupy8"

Of this period auricular systole occupies1"
„ „ diastole „7"
„ ventricular systole „3"
„ „ diastole „5"

During the first period of ventricular systole—the period of *rising tension*—all the valves are closed and the ventricle is getting up pressure. This period lasts .02"-.04". The second period—that of *systolic output*—lasts .2". The intra-ventricular pressure may rise or fall during the latter part of this period, according to the state of the peripheral resistance. The typical impulse curve resembles the curve of intra-ventricular pressure, and exhibits an ascending limb, a plateau and a descending limb. If the carotid pulse curve be recorded, by the tambour method, synchronously with the impulse curve, the time relations can be determined for the human heart. The beginning of the upstroke of the impulse curve marks the commencement of systole; that of the pulse curve marks the opening of the semilunar valves and the beginning of output. The period of rising tension lies between these two points. The beginning of the dicrotic notch indicates the closure of the semilunar valves and the end of the period of systolic output.

The first sound of the heart is synchronous with the upstroke of the impulse curve, and the second sound with the dicrotic notch of the carotid pulse curve.

The maximal systolic pressure exerted by the ventricular muscle varies with the degree of diastolic filling and with the obstruction to systolic output. The heart responds to an obstructed outflow by a greater output of energy, and this it does with no loss of time. The total fluid tension to which the wall of the heart (or an aneurysm) is submitted rapidly increases as the radii of curvature become greater. A heart, when distended, must, therefore, put forth far greater energy in order to raise the contained blood to the pressure of that in the aorta. Hence a dilated heart fails to empty itself and hypertrophies. By its reserve power a vigorous heart may throw out three or even six times the volume of the normal output per minute, or may maintain its output when the lateral pressure in the aorta is thrice the normal value. The systole of the ventricles, by expelling the blood, increases the negative pressure in the thorax, and so aspirates blood into the right auricle. The intra-auricular pressure becomes negative synchronously with the period of systolic output. The flattening of the dome-like diaphragms formed by the auriculo-ventricular valves, a flattening brought about by the contraction of the papillary muscles, favours the filling of the auricles. The intra-ventricular negative pressure, produced by the rebound of the columnæ carneæ at the beginning of ventricular diastole, is of no importance in regard to the filling of the heart. The

heart cannot fill in diastole unless there be a positive pressure in the veins.

The cardiac muscle expresses the blood out of the coronary vessels

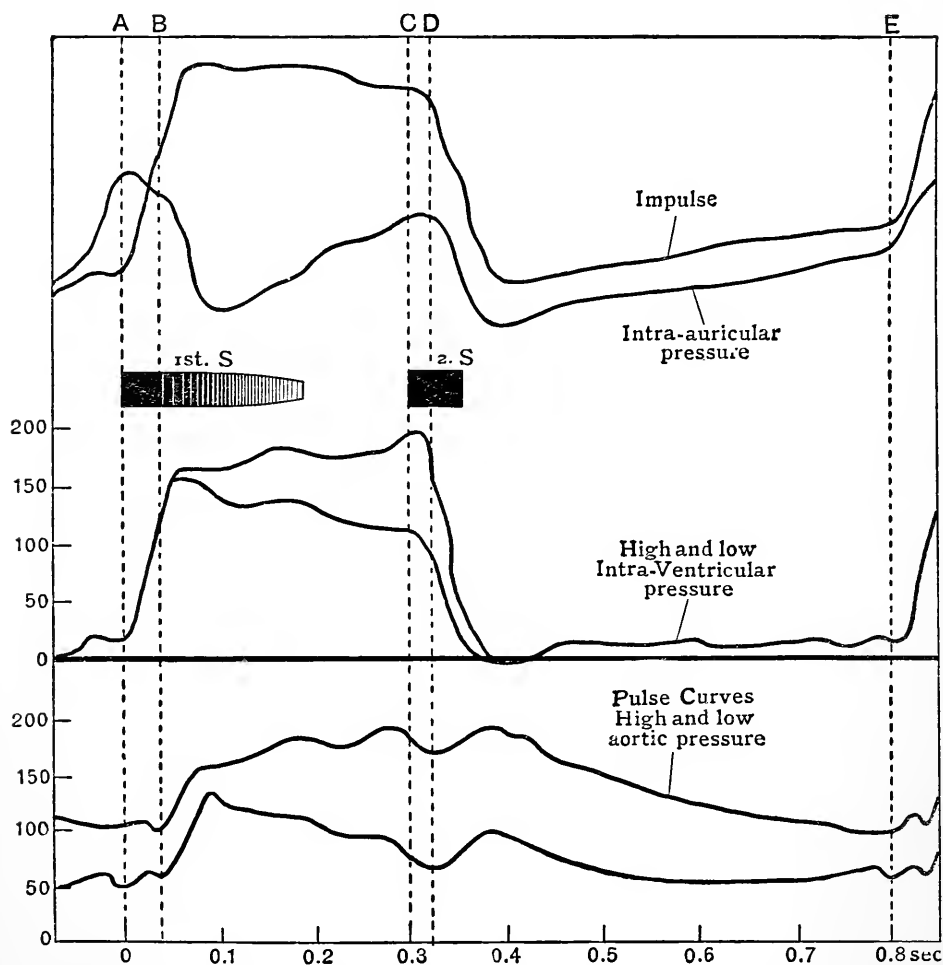


FIG. 17.—The commencement of the impulse curve and of the first sound mark the beginning of ventricular systole (line A); the beginning of the primary pulse wave marks the commencement of the period of systolic output (line B); from A to B is the period of rising tension; the dirotic notch and the second sound mark the tension of the closed semi-lunar valves and the end of the period of output; the waves on the plateau of the ventricular curve are due to elastic vibrations and are partly instrumental; the first fall in the curve of auricular pressure is produced by the systolic output of blood; the blood by leaving the thorax causes a fall in intra-thoracic pressure, and a negative pressure in the auricle, which helps to fill this chamber; the second auricular fall is due to the ventricular diastole; the high-pressure pulse curve is anacrotic. (Modified from Hürthle.)

during systole, and temporarily impedes the velocity of flow in the coronary arteries. It is conceivable, therefore, that the nutrition of the heart may suffer when the heart is accelerated and the period of diastole shortened.

The work of the heart is measured by multiplying the weight of the systolic output by the height of the lift, *i.e.* the mean arterial pressure. The energy spent in maintaining the velocity of flow is a negligible quantity. The output of each ventricle is about 3 ozs. (75-100 grms.), or one-tenth of the body weight per minute. The mean aortic pressure is about 110 mm. Hg. The pressure in the pulmonary artery is about 40 mm. Hg. The work of the heart can be taken as about 700-1000 kilogrammetres per hour. It is estimated that the heart requires about one-fiftieth of the total food value—2500 Calories—required per diem by a resting man.

Sounds of the heart.—*The first sound* is compounded of many tones derived from the sudden tension of the ventricular muscle, the auriculo-ventricular valves and the chordæ tendineæ. It may be augmented by the stroke of the heart against the wall of the thorax. The tension must be sudden and sharp in order to produce an audible sound, and hence it comes about that the first sound is accentuated when the heart is beating rapidly and is only moderately filled, as in conditions of mitral stenosis and after severe hæmorrhage. On the other hand, the muscular element disappears from the sound when the heart is exhausted, as in typhoid fever, empty, as in fainting, or so dilated that the beat is slowly executed.

The second sound is produced by the sudden tension of the closed semilunar valves and is enforced by the vibration of the walls of the aorta and pulmonary artery. The tone varies with the tension, and this is especially so in the case of the extensile and elastic pulmonary artery. Accentuation of the pulmonic sound denotes increased pressure in the pulmonary circulation. Normally the ratio between blood pressure in the pulmonary artery and right ventricle equals the ratio between blood pressure in aorta and left ventricle. Thus the aortic and pulmonic valves close at the same time. Pathologically, as in distension of the right heart from too violent exercise, the ratios become so altered that the sounds are separately heard. The time interval between the two tones must be greater than .04" in order that they may be separately sensed. The sounds of the heart can be recorded by a microphone put in circuit with a capillary electrometer.

Murmurs in the vascular system are occasioned by eddies setting some part of the membranous wall or valve-flaps in vibration. The blood acts as a bow, and the membranous wall as a fiddle-string. On compressing an artery with a stethoscope, eddies are set up as the blood flows, through a narrow orifice, from the part compressed to the more dilated part beyond. The wall of the vessel, in consequence, vibrates. The less the peripheral resistance, the greater is the difference in pressure between the two parts, and the louder the bruit. Uterine souffles, aneurysmal bruits, and venous hums arise in a similar manner. A mere irregularity in the wall of a vessel will not occasion a bruit, for

the layer of blood that wets the wall is motionless. Systolic murmurs are sometimes audible in the hearts of anæmic individuals. Such murmurs can be experimentally produced by the intravenous injection of normal saline solution. Similarly in anæmia the blood is both less viscous and increased in amount, while, owing to lack of muscular tone, the peripheral resistance is lessened. The increased velocity of output may, under such conditions, throw the walls of the aorta and pulmonary artery into audible vibration.

THE PULSE

The radial artery is surrounded by *venæ comites*, the total cross-sectional area of which is greater than that of the artery. To the finger both the breadth of the pulse and the apparent diameter of the radial artery are increased by distension of the *venæ comites*. Thus a feeble pulse, full between the beats, signifies high venous rather than high arterial pressure. The pulse wave is recorded by the sphygmograph (Dudgeon's is the most suitable). The curves are distorted by the instrumental momentum. The instrument should be applied so as to obtain the maximal excursion. We possess no scale by which we can measure the pulse curves obtained by the sphygmograph, and since the form of the curve depends not only on the energy of the heart, but on the peripheral resistance, the turgescence of the *venæ comites*, and possibly on the reflexion of waves from the periphery, its interpretation is exceedingly difficult. The form of the pulse curve may, nevertheless, indicate certain pathological conditions. The primary or percussion wave is a wave of distension, which, taking its origin from the systolic output, travels down the arteries at the rate of about 7 to 8 metres per second. The secondary or dicrotic wave is due to the elastic recoil of the expanded aorta. The wall of the aorta, when thrown into tension, oscillates like an elastic cord. The dicrotic wave is most marked when, with a vigorous heart, the arteries are elastic and the arterioles dilated. Such are the conditions in a young man after a hot bath, or in the first stage of a sthenic fever. One or two smaller oscillations may follow the dicrotic wave. A predicrotic wave is sometimes present. This wave occurs on the plateau of the intra-ventricular pressure curve, and is perhaps due to the reflexion of the primary wave from the periphery. The predicrotic wave may occur on the ascending limb of the pulse curve (anacrotic), and is then a sign of high tension, obstructed outflow, and sustained systole. By compression of the abdominal aorta, the carotid pulse can be made to yield an anacrotic wave. The cerebral pulse curve recorded from the fontanelle of infants is marked by an anacrotic wave, due (probably) to the reflexion of the primary wave from the cranial wall. A forced expiration taken during the record of the pulse curve raises the line of the tracing. This is due to distension of the

venæ comites of the radial artery. Similarly inspiration lowers the line of the curve. The chief points to be determined by the finger in regard

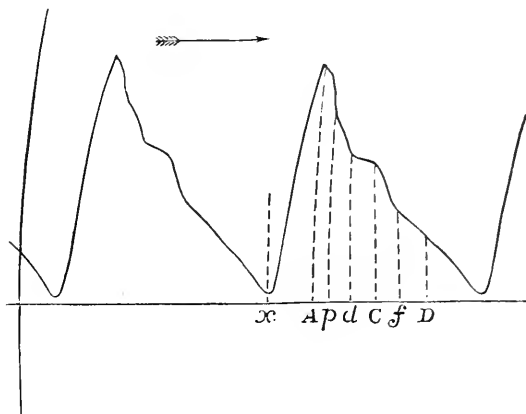


FIG. 18.—Normal radial pulse curve. (After Foster.) *A*, primary wave; *p*, predicrotic notch; *d*, dicotic notch; *C*, dicotic wave; *f*, *D*, post-dicrotic notch and wave.

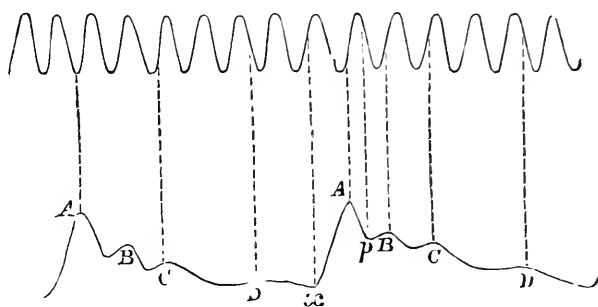


FIG. 19.—Normal pulse curve. (After Moens.) *x*, beginning; *A*, crest with time relations in $\frac{1}{10}$ sec. of primary wave; *p*, predicrotic notch; *B*, predicrotic wave; *C*, dicotic wave; *D*, succeeding secondary wave.

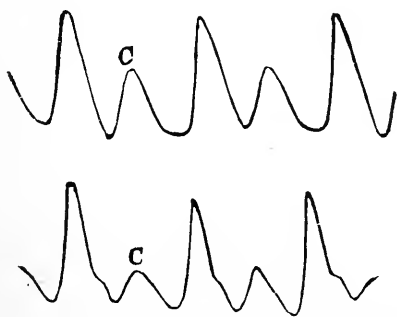


FIG. 20.—Pulse curves with very marked dicotic waves, *C*. (After Foster.)



FIG. 21.—Anacrotic pulse curve (case of aneurysm). (After Foster.)

to the pulse are—(1) the fulness of the swelling in the radial sulcus, composed of the artery and venæ comites; (2) the pressure required

to obliterate the pulse ; (3) the condition of the arterial wall, determined by rolling the artery on the bone ; (4) the frequency, regularity, amplitude, duration, dicrotism of the pulse wave. The daily variations in pulse frequency are very small in a man confined to bed. Warmth and hot food accelerate the pulse. In an air-bath at 65° C. the frequency may reach 160. Change of posture has an important influence. The average frequency is 78 in the standing posture, 70 in the sitting, and 66 in the horizontal position. After prolonged confinement to bed and debilitating disease, the pulse may accelerate 30 to 50 beats when the erect posture is assumed. This is due to the determination of the blood to the lower parts, which, in its turn, results from the lack of muscular tone. The heart accelerates to compensate for the influence of gravity. Under such conditions there is imminent danger of syncope. The change of pulse frequency synchronous with change of posture is a valuable guide to the condition of tone of the muscular system.

Capillary pulse.—The pulse may reach the capillaries in states of vaso-dilatation, of angio-sclerosis, or arterial rigidity, or when the capillaries are filled only at each systole, as in the pink of the nail when the arm is held up above the head, and in cases of aortic regurgitation. Normally the pulse dies away in the ramifications of the arterioles.

A venous pulse may be detected in the jugular vein. The curve of the venous pulse exhibits oscillations synchronous with the auricular systole, and with the systolic output. The pulse is masked by respiratory oscillations, which are excessive in cases of cardiac dyspnoea.

THE ARTERIAL SYSTEM

In the larger arteries there is a soft bed, composed of fine connective tissue and elastic networks, which separates the endothelium from the elastic membrane of Henle. Over-growth of connective tissue in this—the internal coat—produces the condition known as sclerosis. Such over-growth results from any impediment to the velocity of blood-flow, such as is produced by ligation of an artery or frequently repeated and prolonged muscular efforts, *e.g.* the lifting of heavy weights. The middle coat consists, in the smaller arteries, of a thick ring of smooth muscle ; in the larger arteries, of elastic laminæ united by bridges of elastic fibres. Between the elastic laminæ there lie smooth muscle cells and white fibrous tissue. The external coat of the arteries is composed of white fibrous tissue and elastic fibres. The latter are disposed longitudinally. The vasa vasorum do not penetrate beyond the external coat, but white blood corpuscles may wander from these to the internal coat. The vaso-motor nerves end in a plexus of fibrils among the muscle cells. Ganglion cells occupy the larger nodes of the nerve plexus.

The ends of a torn artery retract, coil up within the external coat, and so prevent hæmorrhage. The arteries constrict on the local

application of mechanical irritation, or when emptied of blood. Contraction produced by such means is followed by paralytic dilatation, and hence arises the danger of secondary hæmorrhage. The elasticity of the arteries is submitted to strain seventy times a minute throughout the years of a long life. No inanimate substance would stand successfully so severe a test. The elasticity of the arteries (that is their power to recover their original diameter after distension) is diminished by overstrain, by all wasting diseases, and in chlorosis. The arteries are most distensible (by equal alterations of pressure) at pressures approximating to the normal blood pressure. By exciting constriction of the splanchnic area of arterioles the diameter of the carotid artery may be doubled, and in such case the volume of blood in the arterial system is quadrupled. Small changes in pressure may therefore produce considerable variations in the volume of blood in the arterial system. Pressures above the normal arterial pressure distend the arteries by rapidly diminishing amounts. The distensibility is checked by the white fibrous tissue in the external coat.

The mean arterial pressure in man can be measured by the Hill-Barnard sphygmometer. The instrument consists of an armlet, a spring manometer and a bicycle pump. The armlet is formed of a stiff leather band, on the inside of which there is fastened a flaccid india-rubber bag. The bag is connected by a T tube to the manometer and the pump. The armlet is strapped round the upper arm and the pressure is raised within the bag until the pulsation of the manometer index becomes of maximal excursion. The pressure at which this occurs is the mean arterial pressure. Since the arterial pressure varies with the position of the limb, it is necessary to uniformly measure the pressure with the arm placed on the same level as the heart. A simple form of this instrument has been contrived by which the pressure can be measured both in the radial artery and in a vein. The normal arterial pressure in young (resting) adults is 100 to 110 mm. Hg. During emotional excitement or muscular exertion, the pressure may be 120 to 140 mm. Hg. The pressure falls after muscular exercise and is modified both by this and massage and by baths. Such means can be used with great advantage to lower the peripheral resistance. During sleep or recumbency the pressure equals 95 to 105 mm. Hg. The arterial pressure, from day to day, remains as constant as the temperature, and like the latter cannot safely fall much below normal. In renal disease the arterial pressure is as a rule considerably raised; while in the last stage of Addison's disease a pressure of 70 mm. Hg. has been recorded.

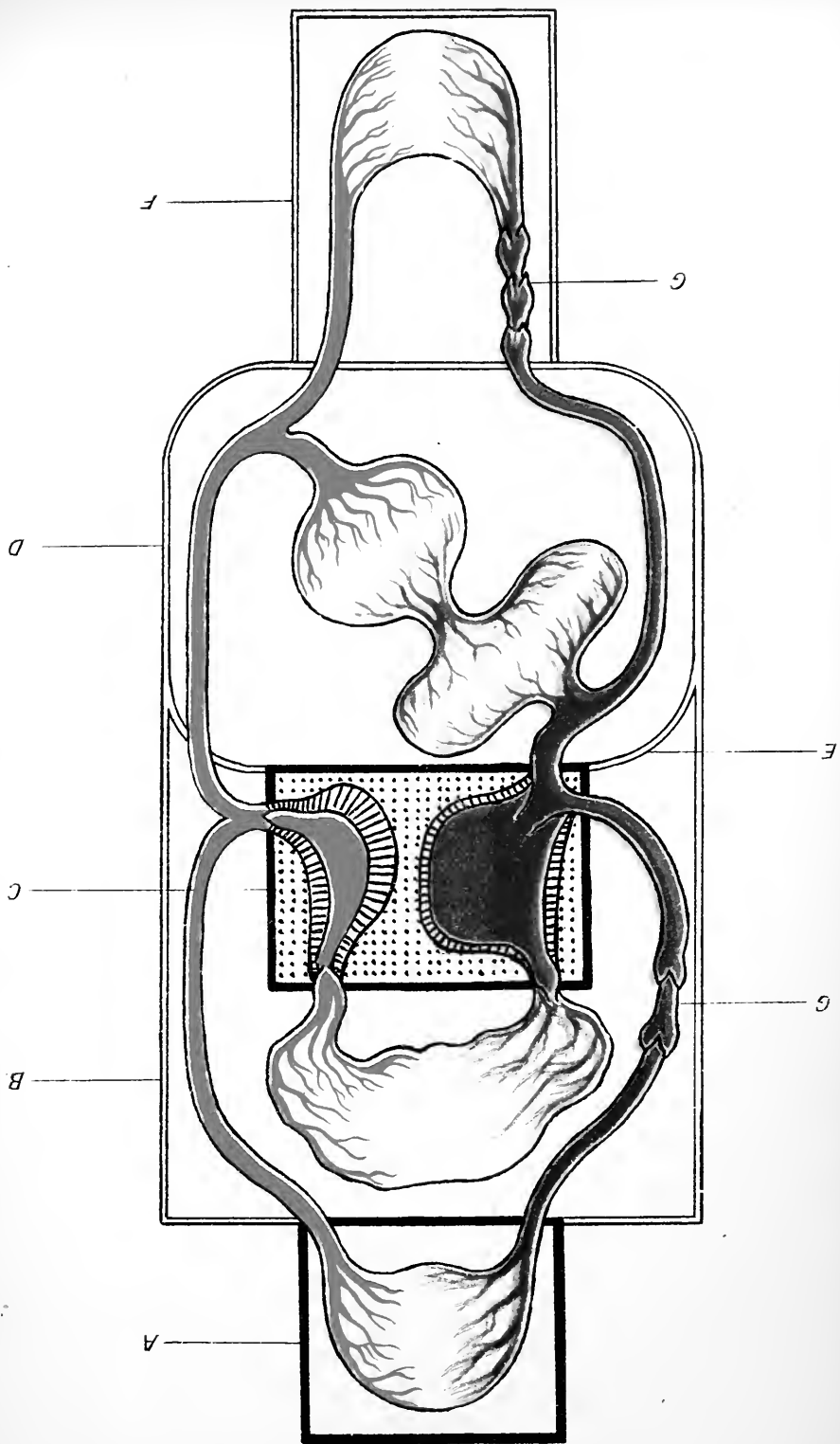
Influence of gravity—Hypostasis.—The position of the body affects the pulse frequency and the amplitude of the cardiac sounds, and is of obvious importance in connection with diseases of the heart and lungs, and in the causation of hypostatic pneumonia, thrombosis, piles, varicose veins, etc., as well as in the relief of syncope, shock and anæmia.

In a man, standing 6 feet high, the hydrostatic pressure of a column of blood, reaching from the vertex to the sole of the foot, is equal to 140 mm. Hg. Nevertheless the circulation remains efficient not only in the horizontal posture, but when the man is either standing erect, or, as a gymnast, is ceaselessly shifting his position. If a model of the circulatory system made of distensile tubing be raised from the horizontal to the erect posture, the lower parts become distended under the weight of the column of fluid, while the upper parts empty and collapse under the atmospheric pressure. In man the arteries and veins of the lower limbs are stouter and more muscular than in other parts, and the venous column of blood is broken into segments by valves. The intra-cranial, vertebral, and azygos veins and the inferior cava are, however, unvalved, and present a long and unbroken column of blood. The heart in man is slung upwards by the cervical fascia and is, during change of posture, restrained both from shifting and from over-dilatation by the pericardium. The patency of the vena cava inferior, at the point where it pierces the diaphragm, is maintained by the muscle fibres of the latter, which pull upon it from all sides. It is protected from the weight of the liver by the spigelian lobe, which rests upon the vertebral column and forms a tunnel for the vein. The intestines are slung upwards to the spine by the mesentery. The liver is slung to the diaphragm, and the central tendon of the diaphragm to the pericardium, and so to the deep cervical fascia. The abdominal muscles are arranged to act like an abdominal

DESCRIPTION OF PLATE III

Diagram of the vascular system showing the cerebral vascular system enclosed by the rigid cranial wall A; the pulmonic system and the heart enclosed by the extensile thoracic wall B; the right and left sides of the heart enclosed by the inextensile pericardium C; the splanchnic vascular system enclosed by the muscular wall D; the diaphragm E separating the abdominal and thoracic chambers; the vascular system of the limbs enclosed by the muscles and skin F; G, the valves in the veins.

In the vertical head-down position the pericardium prevents over-distension of the heart, and the cranial wall limits the congestion of the brain. In the vertical head-up position the muscular and cutaneous walls of the abdomen and limbs prevent the dropping of the blood into the dependent parts, and, aided by the valves in the veins, help to return the blood to the heart. The descent of the diaphragm in inspiration not only aspirates the blood but forces blood from the abdominal organs into the right side of the heart. In inspiration the expanded lungs take more blood, in expiration less. The expiratory diminution of the pulmonic system, aided by the pulmonary semi-lunar valve, helps to fill the left side of the heart. The thick-walled left ventricle and the aorta are scarcely affected by the respiratory changes in intra-thoracic pressure.



belt, and support in an upward direction the weight of the abdominal organs. The capacious abdominal veins are supported under the hydrostatic pressure of the blood by the tense abdominal wall. The tone of these muscles and therefore the capacity of the veins depends upon the nervous system, and especially upon the respiratory centre. The veins in the limbs are supported by the elastic tone of the skin on the one hand, and the tone of the skeletal muscles on the other. The skeletal and visceral muscles act as pumps to the venous side of the system, and with the aid of the valves return the blood to the heart. When man is immobilised in the horizontal position the heart can complete the circulation aided by the respiratory muscles alone, and the muscles can then be relaxed, as in sleep. In the erect posture, on the other hand, the tone of the skin and skeletal muscles is absolutely necessary in order that the circulation may continue. The immobilisation (in the erect posture) of animals with flaccid abdomens, such as tame rabbits, speedily produces cerebral anæmia and death.

The abdominal veins are capacious enough to hold the whole of the blood, and, as the latter sinks, the heart no longer becomes filled in diastole. By either compressing the abdomen with an abdominal bandage, or by immersing the animal up to the neck in a bath of water, the syncope is prevented. In the case of the bath the hydrostatic pressure of the water outside balances that of the blood within, while the bath at the same time, by causing the intestines to float up, prevents the kinking of the portal vein and vena cava inferior. A wild rabbit with taut abdominal wall, and a man "hardened" by exercise are equally immune to prolonged fixation in the erect posture. The toneless, wasted neurasthenic, and the indolent, over-indulgent man "of full habit," on the other hand, are driven to seek relief from hypostatic congestion in massage, abdominal belts, and the bath-treatment. The continued bath-treatment of typhoid fever, by supporting the veins and capillaries, prevents hypostatic congestion and thrombosis; similarly a bath, by equally supporting the blood-vessels of a limb, relieves the pain of inflammatory congestion. Occupations involving the standing or sitting posture for many hours a day tend to produce varicose veins. The immobilisation of a fractured leg in a splint leads to œdema of the foot.

In man the effect of gravity is compensated for, since the vascular system in the erect posture becomes as a whole less distensible. The compensation is brought about with scarcely any change in pulse frequency, and is to be ascribed partly to the increased tone of the skeletal muscles and consequent tenseness of the skin, partly to the more vigorous action of the respiratory pump. So long as this is the case, the hydrostatic pressure of the column of blood in the veins balances that in the arteries, and the energy of the heart continues to act as the driving force. In states of fear, exhaustion, shock, and severe anæmia, the conditions are quite otherwise. Then the tone of the

skeletal muscles is weakened and the patient's knees give under him. The elasticity of the skin and arteries after exhausting disease is greatly diminished, the subcutaneous fat wasted, and the vaso-motor control enfeebled. The permeability of the capillaries may, moreover, be increased. Owing to such changes the blood sinks to the most dependent parts and the aortic pressure falls in the erect posture. The heart then accelerates greatly, and the respiratory pump may be excited to a gasping activity, owing to anæmia of the spinal bulb. Syncope produced by the erect posture is immediately relieved by the assumption of the horizontal posture.

In the conditions of chloroform syncope or asphyxia the right heart is dilated and engorged with blood. The blood can be emptied into the abdominal veins if the patient be sat upright and the thorax be rhythmically compressed. After this treatment has been continued for a few seconds the patient should be returned to the horizontal posture and artificial respiration continued. Inversion increases the paralytic dilatation of the heart under such conditions, and is contra-indicated.

THE CAPILLARY CIRCULATION

The diameter of the capillaries varies from 15 to 4μ . The elastic red corpuscles become temporarily distorted in passing through the narrower capillaries. The spindle-shaped endothelial cells which form the capillary walls exert an anti-coagulant power on the blood. The blood coagulates so soon as it comes in direct contact with the tissue lymph. The branches of connective-tissue cells are here and there cemented to the capillary wall, and these, it is supposed, maintain the patency of the capillaries. After the subcutaneous injection of fluid, the capillaries are not obliterated by the rise of the fluid-pressure in the tissues, but absorb the fluid. There is some evidence to show that the endothelial cells of the hepatic capillaries are capable of protoplasmic movement and of phagocytosis.

The capillary blood pressure stands in closer relationship to the venous than to the arterial pressure. For example, a rise of pressure in the vena cava equalling 10 mm. Hg. raises the hepatic or cerebral capillary pressure 10 mm. Hg., but a similar rise in the arterial pressure has an insignificant effect, for most of the pressure is spent in overcoming the resistance in the arterioles. The capillary pressure and the velocity of the capillary blood flow continually vary owing to alterations in hydrostatic pressure, the pressure of the body against external objects, the contraction of the muscles, the influence of the vaso-motor nerves, the action of the respiratory pump which furthers the return of the venous blood. When the capillary flow is unimpeded the capillary pressure is one-fourth to one-third of the arterial pressure. The capillaries in the foot of a man standing 6 feet high are exposed to a hydrostatic pressure of 140 mm. Hg. The cerebral capillaries would be exposed to the same pressure if

the man stood on his head. In states of complete venous obstruction the capillary pressure rises to that in the aorta. Thus, if all the veins of the leg were obstructed, the capillary pressure in the foot might attain to a height of $110 + 140 = 250$ mm. Hg. Similarly a very considerable rise of cerebral capillary pressure occurs when a man, in bending down to button his boots, lowers his head and at the same time compresses his abdomen. The healthy capillaries can withstand a considerable lateral pressure owing to their minute width, for the circular tension of the wall equals the lateral pressure multiplied by the radius of the capillary. In a capillary tube the velocity of outflow is proportional to the height of the pressure head and to the square of the diameter of the tube, inversely proportional to the length of the tube. The average velocity of flow in the capillaries is 1 mm. per second. A certain velocity of flow is necessary for the nutrition of the capillary wall.

The red corpuscles occupy the axis, and the white corpuscles the peripheral plasmatic layer of the capillary stream. In states of vasodilatation produced by slight local irritation the flow becomes short circuited through the dilated vessels. The white corpuscles pass out through the cement substance—the most permeable part of the wall—of the longer and more tortuous capillaries, where the flow slackens. The emigrated corpuscles follow the lines of flow, and are driven by the transuded lymph from the capillaries to the lymphatic spaces. If the gaps in the cement substance be much widened minute hæmorrhages occur, and the transuded fluid becomes increased in amount and richer in proteid, owing both to the increased pressure in the dilated vessels, and the increased permeability of the capillary wall. The application of a stronger irritant produces stasis, because the plasma entirely escapes and the capillaries become blocked with corpuscles. If the injury be not too severe the plasma may once more enter the capillaries and the corpuscles again begin to circulate. After the obstruction of an artery collateral pathways are in most parts rapidly formed, for the anastomotic capillaries, stimulated by the increased blood-flow, develop into arterioles and arteries. An embolus lodging in a branch of the splenic artery excites long-continued spasm of the anæmic part. When the spasm ceases the veins and capillaries become engorged with the blood which enters through collateral pathways, and extravasation and stasis (red infarction) result. In the brain and kidney the plasma transudes from the capillary area supplied by the embolised artery and a white infarction results.

THE RELATION OF THE LYMPHATIC TO THE VASCULAR SYSTEM

About 75 per cent of the tissues, excluding the fat and skeleton, consists of water. The quantity of blood in the body, according to the latest estimations, equals about one-eighteenth to one-twentieth of the

body weight; that of the lymph is unknown, but it must be great, perhaps two or three times that of the blood. The transudation of lymph from the capillaries depends on (1) the capillary blood pressure (see p. 308); (2) the permeability of the capillary wall. The living cells control both transudation and absorption in such a way as to cause the passage of substances in solution in a direction contrary to osmotic force. This fact is demonstrated by the study of the phenomena of absorption of food and the secretion of urine. The permeability of the capillary wall varies in different parts of the body. Thus liver lymph contains 6 to 8 per cent proteid; intestinal lymph, 4 to 6 per cent; limb lymph, 2 to 3 per cent. Cerebro-spinal fluid may contain 1 to 1.5 per cent; subcutaneous exudations, 5 to 6 per cent; peritoneal exudations, 11 per cent; pleural exudations, 18 per cent. The permeability of the capillary wall is increased by ischæmia, as in states of venous congestion; by poisons, such as snake-venom, extracts of crabs or of mussels, or the toxins of infectious diseases; by scalding and vesicants.

The flow of lymph is maintained by the activity of the muscular system. No lymph flows from the limbs when the body is immobilised, while lymph continues to flow from the abdominal organs, owing to the respiratory movements and the peristalsis of the viscera.

Owing to the potential capacity of the venous system, and the permeability of the capillaries, the arterial pressure cannot be raised by plethora much above the normal. If great quantities of liquid be consumed, or blood or normal saline be injected intravenously, even to the extent of 10 per cent of the body weight, there occurs no noteworthy alteration in arterial or venous pressure, for the extra fluid is accommodated in the veins and tissue spaces, and is, at the same time, rapidly excreted by the kidneys and alimentary canal. If the injection of fluid be carried out rapidly and continuously the liver becomes swollen, the lungs and abdominal organs œdematous, the bladder, alimentary canal, and, finally, the subcutaneous tissues distended with fluid. After the injection of saline the blood-flow is markedly accelerated, owing to the lessened viscosity of the blood and vaso-dilatation. About 80 per cent of the transfused saline transudes from the vascular system within an hour of the injection, and this continues until the blood reaches the normal percentage of solids. Hence it results that the intravenous injection of normal saline has little permanent value in cases of cholera, and after dangerous hæmorrhage. The injection of saline is far more effectual if made subcutaneously and continued for several hours. The injection of animal blood is not permissible, as it leads to hæmolysis.

Blood may be withdrawn slowly, to the extent of 4 per cent, rapidly, to the extent of 2 per cent of the body weight, without lowering the arterial pressure. Compensation is brought about by the contraction of the arterioles and the absorption of fluid from the tissues into the blood.

The absorption takes place synchronously with the hæmorrhage, and thus the blood as it is withdrawn becomes more watery. The injection of a hypertonic solution of salt solution into the peritoneal cavity increases transudation, renders the blood less watery and more viscid, and so diminishes the velocity of blood-flow.

THE VENOUS CIRCULATION

This is impeded by (1) a diminution of cardiac efficiency, *e.g.* valvular incompetence or stenosis, aortic obstruction; (2) obstruction of the filling of the heart, as in cases of pericardial effusion—an intra-pericardial pressure of 15 to 20 mm. Hg. completely obstructs the circulation; (3) obstruction of the pulmonary circulation. The latter may be brought about by spasms of coughing, *e.g.* impaction of a foreign body in the wind-pipe or straining with the glottis closed, by pleuritic effusions and extensive destruction of pulmonary tissue.

The results of general venous congestion are—constriction of the arterial system, hyperæmia of the venous system, a fall in the pressure gradient and consequent diminished velocity of blood-flow, increased capillary pressure, œdema, cyanosis, and a fall of cutaneous temperature. The circulation can only be maintained so long as the right and left ventricle deliver equal volumes of blood. If the left ventricle could continue to deliver 1 c.c. less at each beat there would be “pooled” in the lungs, within one hour, 4000 c.c. of blood. So soon, however, as the left ventricle fails to maintain the full systolic output it ceases to receive the full auricular input, and, in consequence, the pulmonary pressure rises. This in its turn obstructs the output of the right heart, and produces general venous congestion. The equilibrium between the output of each ventricle is thus re-established, but the output is lessened and the arterial supply diminished, for part of the blood is “pooled” in the lungs and venous system. Compensation is brought about by a reversal of the previous inequality of output. Increase of diastolic pressure leads to dilatation of the heart, and this to compensatory hypertrophy. It is obvious that syncope may result when, owing to sudden relaxation of muscular tone (as in fear), the left ventricle discharges the blood at a rate greater than that with which the right ventricle fills. In such case the blood becomes “pooled” in the more dependent parts of the body.

The dyspnœa produced by cardiac insufficiency is due to bulbar anæmia. It aids the circulation, for each respiration pumps the blood from the venous system through the lungs. The panting respiration of a runner is due to “pooling” of the blood in the venous system and dilatation of the right heart. The athlete by training reduces the capacity of the vascular system (hardens himself) and increases the energy of the cardiac muscle.

THE PULMONARY CIRCULATION

The pressure in the pulmonary artery is about one-third the aortic pressure. The four factors which influence the pulmonary circulation are : (1) the systolic pressure of the right ventricle ; (2) the phase of negative pressure in the left auricle ; (3) the pressure exerted by the alveolar air on the pulmonary capillaries ; (4) the diameter of the pulmonary capillaries, which varies with the respiratory expansion of the lungs.

In natural breathing the volumes of air and blood within the lungs vary together, and in the same way. It has been estimated that there is within the lungs during inspiration one-twelfth, and during expiration one-fifteenth of the whole blood quantum. During inspiration the intra-thoracic and intra-alveolar pressures become together lessened ; in consequence the sectional area of the pulmonary vessels increases, while the diastolic filling of the heart is favoured. The blood is not only aspirated into the right heart, but pressed up from the abdomen by the inspiratory descent of the diaphragm. The blood delivered by the right ventricle at first fills up the enlarged pulmonary vessels. It next flows to the left heart in increased volume. As a total result, the pressure in the pulmonary artery falls, while that in the aorta at first falls and then rises. During expiration the conditions are reversed. The intra-pulmonary and intra-thoracic pressures are increased, the sectional area of the pulmonary vessels decreased, the diastolic filling of the right heart lessened. The blood is first expelled by the collapse of the lungs into the left heart and then flows through the lungs in lessened volume. In consequence, the pulmonary arterial pressure rises, while the aortic pressure at first rises and then falls.

The typical effects of respiration are to be observed only when the respiration is slow and deep. When the breathing is rapid, the first effects due to the expansion and collapse of the lung only have time to act, and these respectively lessen and increase the supply to the left heart. In such case the aortic pressure rises during expiration and falls during inspiration.

The distensibility of the pulmonary vessels is so great that three-fourths of the total sectional area of the pulmonary arteries may be obliterated without lowering the aortic pressure.

Artificial inflation of the lungs acts quite otherwise to normal inspiration. When the lungs are artificially blown out, as little as one-sixtieth of the whole blood quantum may be found within them. Artificial inflation raises the intra-thoracic and intra-pulmonary pressures, diminishes the calibre of the pulmonary vessels, obstructs the entry of blood into the right heart, and so lowers markedly the aortic pressure. It is owing to this that inflation of the lungs acts badly as a method of resuscitation. Rhythmic compression of the thorax, on the other hand, not only aerates the lungs, but favours the circulation. If the abdomen be first

gently compressed, the right heart is filled, then by compression of the thorax it can be emptied on through the lungs into the left heart. Thus a pressure of blood can be maintained in the aorta even when the heart has altogether ceased to beat. The supply of fresh aerated blood to the coronary arteries brought about by these means in some cases resuscitates the heart, while the respiratory centre, owing to too prolonged an anoxæmia, remains hopelessly paralysed. In other cases, as often in chloroform collapse, the respiratory centre is aroused to activity by the circulation thus artificially maintained while the heart remains in a condition of paralytic dilatation.

To prevent congestion of the right heart and lungs, the glottis is closed and the intra-thoracic pressure raised during all severe muscular strain. At the same time the fixation of the thorax allows the forcible action of the body muscles.

The existence of constrictor vaso-motor nerves has been determined in the lung, but their influence is by no means strong.

THE CEREBRAL CIRCULATION

The existence of vaso-motor nerves has now been certainly determined in all the organs, including the lungs, pancreas, and liver, with the sole exception of the brain. In this organ, nerves have been demonstrated on the pial vessels, but no experimental evidence of their action has been obtained. The circulation of the brain is somewhat peculiar, since this organ is enclosed in a rigid and not extensile covering. The limbs or viscera can expand when the blood pressure rises, but not so the brain. By the expression of venous blood from the pial veins and sinuses, and the absorption of cerebrospinal fluid, the brain can receive a larger supply of arterial blood when the aortic pressure rises. Similarly, during times of venous congestion, the brain is filled with more venous blood at the expense of the arterial supply. Increase in arterial pressure does not compress the cerebral capillaries and veins, but the velocity of blood-flow is increased, for the circulatory system becomes under these conditions more like a scheme of rigid tubes.

The intra-cranial tension or pressure of the brain against the skull-wall is purely circulatory in origin. It is the same as the cerebral capillary or venous pressure, and varies with every change of aortic or vena cava pressure.

The capillaries in the brain, as in all parts of the body, follow the changes in venous pressure much more absolutely than the alterations in aortic pressure. This is so, because the peripheral resistance lies between the arteries and capillaries. The brain expands in the infant's fontanelle with each expiratory rise of venous pressure. The cerebral pulsation is lessened by any increase of intracranial pressure.

A blood clot, depressed bone, or abscess locally raises the intra-

cranial tension by obliterating the capillaries and raising the pressure at the seat of compression to arterial pressure. In the anæmic area the osmotic pressure of the tissue is increased and secondary œdema thus results. A tumour or area of inflammation, in which the capillary vessels are dilated and the blood-pressure higher than in other parts of the brain, produces compression and anæmia of other parts.

Since the brain is a viscous mass, and the cranial cavity is divided by the falx cerebri and tentorium cerebelli into separate chambers, a local rise of pressure is by no means equally transmitted to all parts. The major symptoms of compression do not arise until the capillaries of the spinal bulb are compressed. Hence a far smaller foreign mass can be endured in the cerebellar than in the cerebral chamber. The cerebrospinal fluid in the meningeal spaces cannot transmit pressure to distant parts, for it is absorbed by the cerebral veins. The outflow of cerebro-spinal fluid, when the cranium is opened, varies with the capillary pressure.

The arterial supply to the brain is so superabundant that the two carotid and two vertebral arteries can be ligatured in the dog at one and the same time, and yet the animal recover perfectly after a short period of paresis and idiocy. The brain in such case receives a supply of blood by way of the anterior spinal artery.

Two of the cerebral arteries can be safely ligatured in one operation in the monkey. To tie one carotid artery in a man with degenerate arteries is not free from risk.

THE PORTAL CIRCULATION

The liver is exceedingly vascular and distensile, and may contain almost one-fourth of the blood in the body. It forms an enormous reservoir for the blood when there is obstruction to the entrance of the blood into the right side of the heart, and the whole of the blood may collect therein if the tone of the splanchnic area of blood-vessels be destroyed and the body be in the erect posture, or if the vena cava inferior be obstructed above the diaphragm. Compression of the liver or vaso-constriction of the portal system empties a large quantity of blood into the right heart, as much as 30 per cent of the total blood quantity. The portal system is supplied with vaso-constrictor fibres from the third to tenth thoracic anterior spinal roots.

When the body is in the horizontal position the pressure in the portal vein equals about 100 to 150 mm. H_2O . This is about the same as the pressure in the cerebral venous sinuses, and may be regarded as the residual pressure after the resistance has been overcome in the capillary vessels.

The portal circulation is largely maintained by the action of the

respiratory pump, the peristaltic movements of the intestine, and the rhythmic contractions of the spleen.

The hepatic capillaries are the most permeable in the body, and a rise of vena cava pressure equal to a very few millimetres of mercury both distends the liver and increases the flow of lymph from the hepatic lymphatics. A small increase in vena cava pressure is also sufficient to drive fluid through the walls of the superficial capillaries of the liver into the peritoneal cavity. The pressure in the hepatic capillaries stands in direct relation to the pressure in the *venæ cavæ*, and obstruction in the right heart or lungs tells back at once on the liver.

THE CORONARY CIRCULATION

The velocity of flow in the coronary arteries is accelerated at the beginning of systole, diminished at the height of systole—owing to the contracting heart compressing the coronary capillaries—and quickened again on diastole. The coronary blood-flow is also quickened by any increase in the force and frequency of the systole. The coronary capillaries can be fed with blood from the coronary veins, and thus these vessels may partly supply a deficiency in the circulation through the coronary arteries. Complete stoppage of the coronary circulation rapidly kills the heart. Occlusion of the descending or circumflex branches of the left coronary artery generally causes arrest of the ventricular contraction. Occlusion of the right coronary artery rarely causes arrest. Fibrillar contraction produced by occlusion of the coronary arteries can be set aside by renewing the circulation. The branches of the coronary arteries are physiologically terminal, *i.e.* the anastomosis is not sufficient to prevent necrosis of the part supplied by the occluded branch.

Vaso-motor nerves are said to reach the coronary vessels from both the vagus and the stellate ganglion.

THE VASO-MOTOR SYSTEM

The tone of the blood-vessels depends on the quality, pressure, and velocity of the blood. The tone of the arteries is controlled by the vaso-motor centre in the spinal bulb. The vaso-motor nerves are of two kinds, vaso-dilator and vaso-constrictor. The portion of the spinal bulb, the integrity which is necessary for the general reflex control of the vascular system, extends from a point 4 mm. above the *calamus scriptorius* to within 2 mm. of the posterior *corpora quadrigemina*. Destruction of this part produces general vaso-dilatation, and a profound fall of blood-pressure; excitation is followed by the converse effects. There are subsidiary centres in the spinal cord, *e.g.* reflex vaso-dilatation of the penis may be provoked after the dorsal cord has been divided. A recovery of tone, depending on the

local neuro-muscular mechanism, follows the stage of dilatation which at first results from section of vaso-motor nerves. The arterioles may be almost entirely obliterated by the spasm of their muscular coat (cf. Raynaud's disease). In rare cases blindness is produced by spasm of the retinal artery. The vaso-constrictor nerve fibres are of small calibre (2 to 4μ) and are the axons of spinal cells, situated probably in the lateral horn of gray matter. They pass in the white rami communicantes, as "præganglionic" fibres, to the sympathetic ganglia, and each fibre has a "cell station" therein. The post-ganglionic fibres (axons of the sympathetic ganglia) are as a rule non-medullated. The vaso-constrictor fibres for the whole vascular system arise from the anterior spinal roots, from the second dorsal to the second lumbar inclusive. The vaso-dilator fibres are not limited in outflow to the thoracic region, and have their cell stations in the ganglia in the neighbourhood of the blood-vessels. Every vaso-motor fibre has one cell station between the spinal cord and its termination.

The head and neck and salivary glands.—Vaso-constrictors from the cervical sympathetic. Origin, first to fifth thoracic roots. Cell station, superior cervical ganglia. Vaso-dilators from cervical sympathetic (to mouth), lingual branch of fifth nerve, chorda tympani (to submaxillary) auriculo-temporal nerve (to parotid gland). The cell station of the vaso-dilators of the mouth is in the superior cervical ganglion.

The lungs.—Vaso-constrictors from the upper thoracic roots, especially third to sixth.

Upper limbs.—Vaso-constrictors from third to eleventh thoracic roots. Cell station, stellate ganglion.

Lower limbs.—Vaso-constrictors from lower thoracic and upper lumbar roots, especially eleventh to third lumbar. Cell stations, sixth and seventh lumbar, and first and second sacral ganglia. Vaso-dilator fibres for the limbs issue by the posterior roots.

Liver, spleen, pancreas, stomach, and intestines.—Vaso-constrictor and dilator fibres from the splanchnic nerves. Origin, thoracic roots. Some of the dilator fibres arise from the vagi. Cell stations for the stomach in the cœliac ganglion; for the small intestine in the superior mesenteric; for the large intestine in the inferior mesenteric ganglion.

Kidneys.—Vaso-constrictors and dilators from the lower thoracic roots. Cell stations in the cœliac and renal ganglia and in the solar plexus.

Generative organs.—Vaso-constrictor from the lower lumbar, and dilator fibres from the second to third sacral roots. Cell stations in the sacral ganglia.

The excitation of an afferent nerve produces, as a rule, general constriction and local dilatation. These changes determine the blood to the part excited. The heat loss is largely dependent on the vaso-motor system, which thus becomes an important factor in the regulation of the body heat.

LEONARD HILL.

DISEASES OF THE CIRCULATORY SYSTEM

DISEASES OF THE HEART

The diseases of the heart and arteries, as subjects of medical study, are not only very important practically but full of genuine scientific interest. Their practical importance is obvious. Cardiac and vascular diseases are accountable for a large percentage of deaths at all ages, and for a still larger proportion of the cases of illness, distress and disablement of ordinary life. This arises from several circumstances. In the first place, acute disease of the heart is very liable to terminate in chronic disease, that is, in permanent damage of the valves, myocardium, or pericardium. Secondly, many of the influences of common life—food, stimulants, mechanical strain and nervous activity—peculiarly affect the organs of circulation, first causing functional disturbances, then, if their actions be prolonged, metabolic disorders, and finally disease of the vessels, myocardium, and valves. Thirdly, the circulation being in particularly intimate physiological relations with all parts, and especially with the blood, lungs, and kidneys, it soon comes to participate or be involved in disease originating in these.

From the purely scientific point of view, diseases of the heart present many attractions. In acute inflammation of the endo- and pericardium, a favourable opportunity is afforded of observing by methods of physical examination the origin, progress, and terminations of morbid processes. In these and in chronic valvular disease are employed most of the methods of physical examination in clinical use; and many of the most interesting physical signs presented by visceral lesions may be observed in this way. Further, in chronic valvular disease is to be seen the most obvious and easily intelligible instance of that great method of physiological recovery which is known as compensation, and with which and its conditions it is necessary to become familiar in order to appreciate the practical problems of successful prognosis and rational treatment.

GENERAL ETIOLOGY

(1) Of the extrinsic causes of disease of the heart, the commonest are those which reach it through the blood, either directly or by the coronary arteries. These are *micro-organisms* and the recognised or

hypothetical *toxines* of the acute specific fevers, especially septicæmia, pyæmia, rheumatism, scarlet fever, diphtheria, measles, influenza, syphilis, and gonorrhœa. The heart at best offers but feeble resistance to these powerful pathogenetic agents, and very often predisposing conditions exist which make it still more readily vulnerable by diminishing natural resistance. Such are childhood, when the cardiac tissues are peculiarly delicate; middle and advanced age, when nutrition is failing from vascular degeneration; previous valvular or parietal lesions, repaired with ill-nourished scar-tissue; and general constitutional deterioration, consequent on imperfect hygiene, sedentary habits, abnormal nervous influences through the vagus and sympathetic, disordered alimentation and deficient elimination. The result is disease, in the form of acute and chronic inflammation of different kinds—endocarditis, myocarditis, pericarditis—and of parenchymatous degeneration. Further, inasmuch as most cases of chronic valvular lesions originate in acute endocarditis, it will be seen how large a part in the etiology of cardiac disease is played by micro-organisms and their products.

(2) Closely related to the group of causes just mentioned are *anæmia* and *toxæmia*, in the most comprehensive sense of the terms, which account for many disorders and diseases of the heart: simple anæmia (with its many causes, including hæmorrhage), pernicious anæmia, leukæmia, scurvy, purpura, etc.; the toxic products of disordered metabolism—uric acid, urea in excess, and their chemical allies (for instance, in gout and Bright's disease); and a number of poisons, whether animal (such as snake poison), vegetable (*e.g.* tobacco), mineral, gaseous, or compounds like chloroform, antipyrin, etc. The pathological actions of all these hæmic and toxic causes of cardiac disease, taken together, in addition to giving rise to simple functional disorders, affect chiefly the nutrition of the valves, the endocardium as a whole, the myocardium, and the pericardium, setting up different kinds of degeneration; but some of them act much more swiftly and specifically upon the neuro-muscular structures of the heart, as poisons proper. It must also be observed that the anæmia and general deterioration which are developed in the course of many cases of chronic heart disease steadily sap its own nutrition; and that thus chronic cardiac disease tends to progress, a vicious circle having been established. A similar pathological effect on the cardiac structures can often be traced to *local anæmia*, consequent on disease of the coronary arteries—syphilitic, degenerative, embolic, etc.—the extent and situation of

the lesion in the myocardium corresponding with the size and distribution of the affected vessel.

(3) A third set of causes of disorder and disease of the heart belong to an entirely different order of pathogenetic influences. These are *physical* in nature. Violence in various forms accounts for a very small proportion of cardiac lesions, including wounds and other injuries, and disablement of the heart by the irruption of emboli, of parasites, and of pathological gatherings in the neighbourhood. More frequently the heart is dislocated, embarrassed, and possibly overwhelmed by the physical effects of fluid or gaseous collections in the pleural and peritoneal cavities, or by distension of the stomach and bowels.

The most important causes of cardiac disease of the physical order require more specific notice. They are of the nature of stress. The systemic arterial pressure acting upon the left ventricle, or the pulmonic arterial pressure acting upon the right ventricle, sometimes reaches such a height as to threaten to strain the walls and valvular apparatus. Under these circumstances there come into action several of the regulating mechanisms which have been already described, p. 307. The myocardium responds to the increased intra-cardiac pressure by more vigorous contraction; the rhythm falls in frequency through the medium of the cardiac centre and vagus, and the dilator mechanism relaxes the arterial walls, permitting easier emptying of the left ventricle. At the same time the highly extensile tissues of the heart permit distension of the chambers and afford temporary accommodation for the excess of blood; the dilatation of the chambers, if it be extreme, even allowing leakage between the ventricles and auricles—"safety-valve action." But these several provisions for preventing strain may fail. Either, on the one hand, the stress is altogether excessive or too sudden, during violent muscular exertion such as racing, and the result is valvular rupture or strain of the muscular walls in a perfectly sound heart; or, on the other hand, even an ordinary muscular effort may have the same unfortunate effect if one or other of the cardiac structures have been previously unsound—the valves diseased, the myocardium degenerated, the general nutrition of the heart unhealthy, or the elasticity of the tissues reduced by age or antecedent strain.

In by far the larger number of instances, however, the physical causes of cardiac disease are not sudden and brief, but slow and persistent in their action: on the left heart, for example, of the labourer or of the subject of chronic Bright's disease or arterial

sclerosis with high arterial tension, and on the right heart of the subject of chronic pulmonary disease, particularly emphysema, fibrosis and chronic pleurisy. Originally sound, the heart in such subjects gradually undergoes enlargement; it is hypertrophied and dilated; the valvular apparatus comes to be the seat of chronic inflammatory and degenerate changes; and, the cause continuing, the morbid condition progresses from bad to worse. Similarly, disease of one set of valves—say, the aortic—will cause disease in another set of valves situated physiologically in their rear, the mitral, by raising the physical pressure on them. In other instances the mechanical stress acts upon the heart *a tergo*. During violent exertion the venous blood is returned in such volume, and at so high a pressure relatively, that the right auricle and ventricle are over-filled, their walls are over-stretched, and embarrassment or even permanent damage results; and this kind of injury also may be either acute or chronic in its development.

(4) An intangible, obscure, often unsuspected, but equally real class of causes of disorder and disease of the heart are of a *nervous* kind. Whilst disturbances of the central nervous system by intrinsic disease, by sensory and emotional influences, and by morbid impressions reaching it from the viscera and suddenly transmitted to the heart are amongst the most familiar causes of palpitation, faintness, and other manifestations of cardiac discomposure, the student is apt to overlook or underestimate the important part played in the production and aggravation of heart disease by mental and reflected impulses of a persistent but less declared character, such as are associated with worry, anxiety, misfortune, and the depression of chronic visceral disease. Not only functional troubles, but structural changes in the walls of the heart may originate, and seriously increase, in this way. Here also, as in connection with nutrition, the personal element is a factor in disease. Some individuals possess peculiarly sensitive hearts, which fall into a condition of morbid “irritability” under the influence of ordinary—that is, perfectly reasonable—excitement or exercise.

(5) The remaining causes of disease of the heart of an extrinsic kind that call for notice are easily disposed of. *Parasites* are found in the cardiac walls, chambers, and pericardium, whether they actively invade these parts, or are carried to them by the blood-current. *New growths* may involve the tissues there, as elsewhere, sometimes of primary, more often of secondary origin.

The extrinsic causes of disease of the heart which have just been enumerated are brought into relation with the heart, favoured

in their action on it, or otherwise assisted, by a number of intrinsic circumstances, different in different individuals. Only the most important of these "predisposing" conditions require to be mentioned.

The male *sex* exposes the heart to mechanical stress, both sudden and sustained; to the high tension, the disposition to coronary degeneration, and the direct taxation of the cardiac walls and nerves inseparable from business and professional work; to the actions of such cardiac poisons as alcohol, tobacco, and uric acid, and the virus of syphilis and of gonorrhœa. The heart of the woman, on the other hand, is heavily handicapped by the many circulatory and nervous burthens connected with menstruation, pregnancy, parturition, the puerperium, lactation, and the menopause; and she is the favourite subject of certain classes of circulatory neuroses, such as pseudo-angina and Graves's disease. Each of the different *ages* of man affords an opening for a particular set of pathogenetic influences to assail the heart—infancy and childhood for the infective fevers; adolescence for rheumatism, chorea, the disturbing effects of educational pressure, simple anæmia, and menstrual irregularities; adult life for syphilis, gonorrhœa, septic diseases, tobacco, physical strain, and nervous excitement and exhaustion; middle life for uric acid in its different manifestations, alcohol (both directly and indirectly), worry and other depressing nervous influences, Bright's disease, and premature degeneration of the arterial walls, particularly the coronary; old age for the circumstances that favour degeneration of the cardiac parietes, of the circulatory system, and of the body as a whole. It is important to realise that, speaking broadly, diseases of the heart, however similar may be the symptoms and signs by which they are clinically characterised, are of an entirely different nature according as they originate in the earlier or in the later years of life. In younger subjects they are always infective or inflammatory, unless in the rare instances of congenital affections to be presently mentioned. In older subjects the results of earlier acute inflammatory disease are naturally met with in large numbers; but the many lesions which commence at this period of life are, with few exceptions, essentially degenerative. The bearing of this fact on prognosis and treatment will appear later on.

If we regard what are essentially the same facts from another point of view, that of *occupation*, we find that this accounts for the incidence of the efficient causes of diseases of the heart in many instances—by necessitating exposure to exertion; by affording

opportunity and offering temptations to indulgence in alcohol, large eating, and other bad habits; by inducing general anæmia and functional disorders of the viscera, as in town life; by establishing morbid conditions of the nervous system, and so on. *Previous damage* powerfully contributes to the invasion and advance of fresh disease, whether of the valves or of the myocardium. A morbid disposition to premature (essential) degeneration of the cardiac walls is sometimes found running in a family, several members of which perish at a comparatively early age of disease of the heart. Incidents of anomalous development *in utero* account for an important group in cardiac pathology—the *malformations*.

GENERAL MORBID ANATOMY AND PATHOLOGY

I. DISORDERS AND DISEASES OF THE VASCULAR SUPPLY OF THE HEART

The first group of morbid changes in the heart produced by the causes, and under the circumstances, described in the preceding section, are those which primarily affect the blood-vessels of the myo-, endo-, and pericardium—that is, the coronary system. *Mechanical congestion* of the cardiac walls is one of the many visceral changes associated with failing heart, venous distension, and dropsy. The myocardium at first is simply œdematous, but it gradually becomes pigmented, dense, fibroid, and atrophied, and serum is effused into the pericardial sac—*hydropericardium*. A far more important disease belonging to this group is *arterial degeneration*, the outcome of chronic arteritis, syphilis, or Bright's disease, inasmuch as it produces various degrees of narrowing of the coronaries up to complete occlusion, and many unfortunate effects on the muscular walls and valves to be noticed presently. With these there is sometimes associated *thrombosis*, ending in complete occlusion of the lumen of one or more branches of the nutritive vessels, and local necrosis, infarction, fibrosis, and possibly rupture of the myocardium. Similar changes are sometimes traced to coronary *embolism*, which may lead to abscess if the embolus be infective. The coronary arteries are also the seat of *aneurysm*, particularly at their mouth in the sinuses of Valsalva.

II. DISORDERS AND DISEASES OF NUTRITION: DEGENERATIONS

I. PARIETAL.—The different kinds of myocardial degeneration are in great measure effects of the morbid conditions of the coronary arteries just noticed; but in other instances they can be traced, not to the quantity, but to the quality of the blood supplied by them, including toxæmia. In *simple atrophy* of the myocardium, which usually involves the entire heart in old and cachetic subjects, the individual fibres are reduced in size and the striation is indistinct, while brown pigment granules are prominent within them. *Fatty degeneration* of the myocardium is a far more important disease, whether it be general, as a consequence of anæmia or toxæmia, or a local effect of coronary disease and impaired nutrition, as just described. Fatty degeneration

of the heart is recognised by its peculiar colour, the yellow of a faded leaf, which is seen distributed as small spots, dashes, or patches beneath the pericardium, or beneath the endocardium of the ventricle, particularly in the muscoli papillares and columnæ carneæ. Sometimes the disease is universal, as in profound anæmia and phosphorus poisoning. Under the microscope, the muscular fibres of these discoloured spots, and of other parts that appear healthy to the naked eye, are seen to have lost their striation, and present instead innumerable highly refracting oil particles, derived from the degenerated tissue-elements.

Local necrosis of the myocardium has been already traced to obstruction of the coronary arteries and their branches. It takes the form of softening and yellowish discoloration of the affected area, possibly with hæmorrhage into, and even rupture of, the muscular tissue, local pericarditis, and endocardial thrombosis, the whole constituting in some instances a hæmorrhagic infarct. Histologically, these soft areas present complete necrosis and fatty degeneration, with breaking down and segmentation of the muscular fibres. In less complete and rapid vascular obstruction, atrophy and fibroid change occur.

Acute granular degeneration of the myocardium is most familiar as a change involving the entire heart in the acute specific fevers, such as pneumonia, diphtheria, and typhoid; but it occurs also as a local process in some of the vascular disorders and diseases already mentioned. The heart is soft and flabby, the walls of the chambers collapsing upon them, instead of standing out firmly on section; the colour is a dirty crimson, or yellowish- or grayish-red, and decomposition readily occurs. Microscopically, the morbid changes are very complex—loss of striation, cloudy swelling, fatty metamorphosis, hyaline degeneration, and vacuolation and segmentation of the muscular fibres.

Calcification of the muscular fibres is a rare pathological change.

Increase of the adipose tissue under the epicardium and between the muscular fibres constitutes *fatty infiltration* of the heart, which must be carefully distinguished from fatty degeneration, in which the individual muscular fibres are metamorphosed, although the two conditions very frequently coexist. The oil-laden connective-tissue cells may increase to such an extent in fat persons as to invade the inter-muscular spaces, compress or separate the fibres, and seriously interfere with their nutrition. The organ is increased in bulk and weight, and its chambers may be dilated.

Fibrosis, whether general or local, is variously related to other

diseases of the myocardium. It is a result of prolonged mechanical congestion of the coronary veins; of incomplete arterial occlusion, degenerative, thrombotic, or embolic; of chronic myocarditis; of the repair of acute local myocarditis, and of neighbouring endocarditis and pericarditis; of wounds, and of parietal or vascular syphilis. *Local* fibrosis is easily recognised as whitish, gray, opaque, or glistening spots, streaks, or patches in the myocardium. The

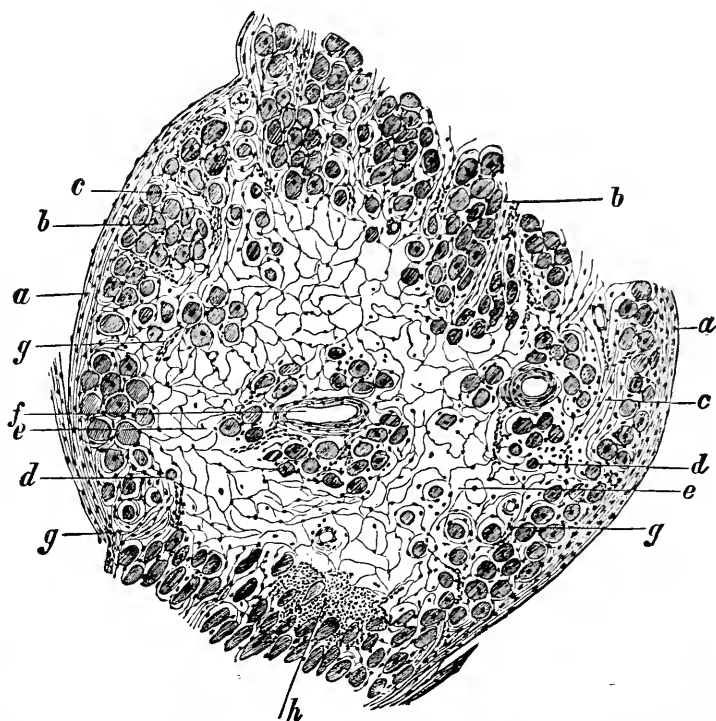


FIG. 22.—Fibroid disease of the heart; transverse section. A columnar carnea which has undergone fibroid change. $\times 40$. *a*, endocardium; *b*, transverse section of normal muscle-cells; *c*, hyperplasia of connective tissue richly infiltrated with cells; *d*, atrophic muscle-cells in the hyperplastic connective tissue; *e*, fibroid tissue without cells; *f*, vein surrounded by a few remaining muscle-cells; *g*, small blood-vessels; *h*, cellular tissue. (Ziegler.)

microscopical characters of cardiac fibrosis are illustrated in Fig. 22. New fibrous tissue is developed by fibrillation from the connective-tissue cells. Abundant leucocytes, which have migrated from the blood-vessels, are seen in association with these in the first stage of the process, but they are believed to take no part in regeneration proper, and they afterwards disappear. The newly formed connective tissue then contracts, narrows the nutrient blood-vessels, and in this way, as well as by direct pressure, it causes atrophy of the muscle-cells and weakness of the walls. When such fibroid

patches yield to the intra-cardiac pressure, and bulge outwards or into another chamber, a condition is produced that constitutes one kind of *chronic aneurysm of the heart*. This is most common near the apex of the left ventricle, contains blood-clot and fibrin, and may rupture into the pericardium or into another cardiac chamber. *General* fibrosis is necessarily of moderate degree. It imparts a firm, tough character to the walls of the heart, and is associated with enlargement.

2. VALVULAR.—*Atheroma of the valves*.—The morbid changes produced in the valves of the heart by chronic disorders and diseases of nutrition, usually vascular or toxic in origin, are commonly known by the comprehensive name of atheroma. The degenerative factors of this complex process are fatty metamorphosis of the endothelial and connective-tissue cells of the valvular tissues; jelly-like degeneration; hyaline and calcareous degenerations, as more advanced changes; and finally softening and ulcerative destruction of the segments. The reconstructive factors of the atheromatous process consist of a very slow fibrosis attending the earlier stages of degeneration; more actively inflammatory and regenerative changes associated with the softening and ulceration, which also end in fibrosis; and the formation on the affected surfaces of thrombi, which gradually become organised. The result is the conversion of the smooth, thin, and delicately fitting valves into rough, thickened, shrunken, and irregular structures, which are stiffened and impaired in their mobility, interfere with the uniform movement of the blood over their surface, obstruct its free passage through the related openings, and by their irregular apposition become incompetent as valvular apparatus.

III. INFLAMMATION

Inflammation of the cardiac structures has been already referred to, as it is variously related to disturbances of the coronary circulation and to nutritional disorders and diseases of the parietes and valves. As a process of independent importance, carditis includes myocarditis, endocarditis, and pericarditis, individually and in mutual association. In the great majority of instances it is the effect of the direct actions of micro-organisms and their products, of toxæmias, or of traumata.

1. MYOCARDITIS.—*Purulent inflammation* of the myocardium is met with in the form of one or more abscesses, bounded by a zone of hyperæmia and infiltration. Streptococci, staphylococci,

pneumococci, and the micro-organisms of different acute specific fevers occupy the spaces between the tissue-elements, which are variously degenerated, and within and around the vessels, which may be thrombosed. *Abscess of the heart* may burst into one of the chambers (*acute cardiac aneurysm, ulceration of the heart*), or into the pericardial sac; or its contents may become inspissated and absorbed, a scar remaining. In other instances myocarditis is *non-purulent* or *parenchymatous*, appearing as yellowish or grayish swollen spots or layers, with a reddish border, soft and friable. Here the muscular elements are variously degenerated, in association with the organisms distributed around, and the connective tissues are the seat of infiltration and proliferation. Acute myocarditis is usually accompanied by endocarditis and pericarditis. In protracted cases myocarditis proceeds to fibrosis and its consequences—*chronic myocarditis, chronic cardiac aneurysm, fibrosis, fibroid disease*.

2. ACUTE ENDOCARDITIS.—Acute inflammation of the lining membrane of the heart is the most common and important of all cardiac lesions caused by infective organisms, their products, and poisons of other kinds, and is especially familiar in connection with acute rheumatism. The valvular endocardium of the left side is its favourite seat, particularly that of the mitral and aortic cusps, and of these not the free edges but the lines of mutual apposition and percussion, and the surfaces immediately exposed to the moving blood, namely, the auriculo-ventricular opening and the cardiac aspect of the aortic segments. But if the cause be virulent, any portion of the endocardium may be attacked, and the morbid process may spread into the substance of the valves, the chordæ, and the myocardium itself (which as we have seen may be involved independently), and produce very severe and complex lesions, including valvular aneurysm, rupture of the valves and chordæ, or acute cardiac aneurysm. Endocarditis is first recognised by opacity and slight swelling of the affected valves, produced by degeneration of the tissues and interstitial infiltration; to these changes there is presently added superficial thrombosis. If the disease be actively infective, the parts necrose, leaving ragged erosions, their bases formed of grayish sloughs and loose thrombi, their edges thickened, undermined and fringed with vegetations; or the vegetations may be so large and abundant as to constitute exuberant masses of soft growth. Portions of these are readily detached and fall into the blood stream, where they lead to septic embolism and its consequences (see *Infective Endocarditis*, Vol. I. p. 77).

More often the constructive factor of the inflammatory process

is in the ascendant, and repair occurs, with the production of vegetations, warty growths, or polypi, varying in size from a pin's-head to a pea; but repair here has often the unfortunate effect of producing adhesions, puckerings, thickenings, irregularities, and other permanent changes in the valves and their apparatus, which render them incompetent; or it ends in stricture (stenosis), with consequent obstruction at the openings. In either event *chronic valvular disease* is the result (Figs. 23 and 24). Of the ulcerative kind of endocarditis, and between this and the plastic or sclerosing kind, there are many varieties of the morbid process, leading to extremely diverse forms of valvular and parietal lesions.

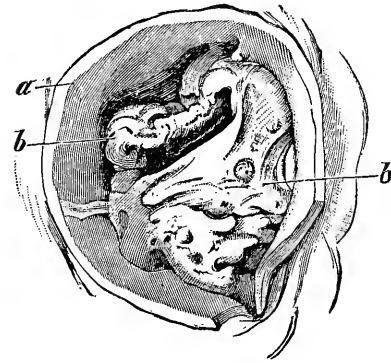


FIG. 23.—Chronic valvular disease. Aortic valves viewed from above; their cusps thickened and occupied with firm, partly organised, partly calcified thrombi. Aortic stenosis. *a*, transverse section of the aorta above the valves; *b*, calcified thrombi, adherent to the sinuses. Nat. size. (Ziegler.)

The microscopical characters of acute endocarditis are in some important respects different from those of inflammation in general,

consequent on the peculiar relations and histological structure of the affected parts. The valvular vegetations prove to consist mainly of laminated thrombi, deposited from the circulating blood upon necrotic areas of the endothelium and subjacent tissues, which have been the first to yield to the pathogenetic influence, whatever its nature. Abundant cells, partly phagocytic exudations from the

neighbouring blood-vessels, partly germinal products of the connective-tissue corpuscles, infiltrate the thrombi and proper structures

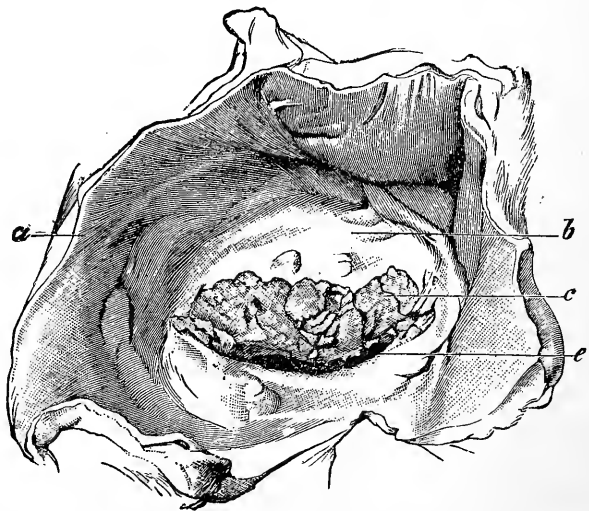


FIG. 24.—Mitral valve viewed from the auricle, the posterior segment thickened and occupied by calcified thrombi. Mitral stenosis. *a*, auricular wall; *b*, thickened posterior segment; *c*, thrombus, partly calcified, partly organised; *e*, mitral opening. Nat. size. (Ziegler.)

of the valves (Fig. 25). Thus the thrombi become organised, and eventually repair is effected by fibrous tissue; but this subsequently contracts, and if the tissues have been deeply or extensively involved, the result is either deformity and incompetence of the valve, or stenosis of the associated ostium.

In the ulcerative disease specific organisms of different kinds are found abundantly invading the superficial tissues and penetrating thence into the deeper structures. The resulting necrosis is accompanied with thrombosis and the other manifestations of inflammatory reaction just described, but these prove insufficient to arrest or limit the destructive process, which leads to disintegration instead

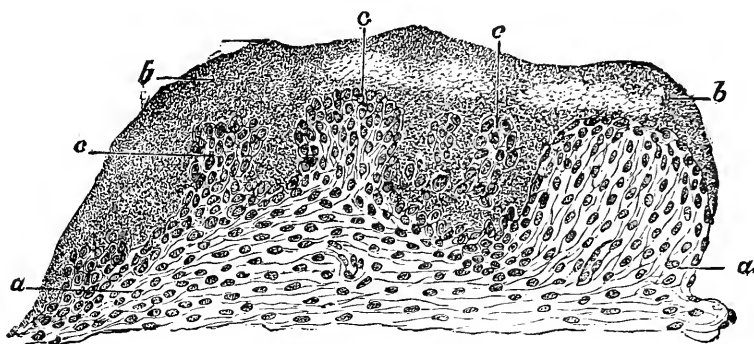


FIG. 25.—Acute endocarditis of an aortic cusp. *a*, hypertrophic connective tissue of the valve, with vessels; *b*, granular thrombi; *c*, fibro-cellular outgrowths of the endocardium. $\times 60$. (Ziegler.)

of fibrous repair of the valves, and occasionally to suppuration of the deeper structures.

3. CHRONIC VALVULAR LESIONS.—Chronic valvular lesions of inflammatory origin are most commonly the result of acute endocarditis, as described under the preceding head. In other instances the valvulitis is chronic, caused by prolonged physical stress. Bright's disease, alcoholism, and gout may sometimes be productive of a slow interstitial endocarditis, but the valvular lesions associated with these morbid influences are far more often degenerative (atheromatous), as already noticed. Certain less virulent kinds of infection also lead to chronic endocarditis.

Chronic inflammatory lesions of the cardiac valves are with few exceptions found on the left side of the heart. The mitral and aortic valves are by far most commonly affected; the pulmonic and tricuspid in less than one per cent of all cases. The morbid changes in the valvular segments fall conveniently into two groups. (1) The valves prove to be *incompetent* when subjected to the

hydrostatic test: *regurgitant* lesions. These are the result of mal-apposition or deformity of the valvular segments, which are the seat of vegetations, irregular polypoid or pendulous bodies, or masses of considerable size along the lines of mutual contact; of sclerotic thickening or puckering; of eversion of part or the whole of their free borders; of laceration; or of local dilatation (valvular aneurysm). (2) The ostia prove to be *stenosed* when tested with the finger or accurately measured: *obstructive* lesions. These are the result of unnatural adhesions of the margins of the segments or cusps; of shortening and fusion of the chordæ tendineæ; of the presence of polypoid masses projecting into the ostia; of fibroid

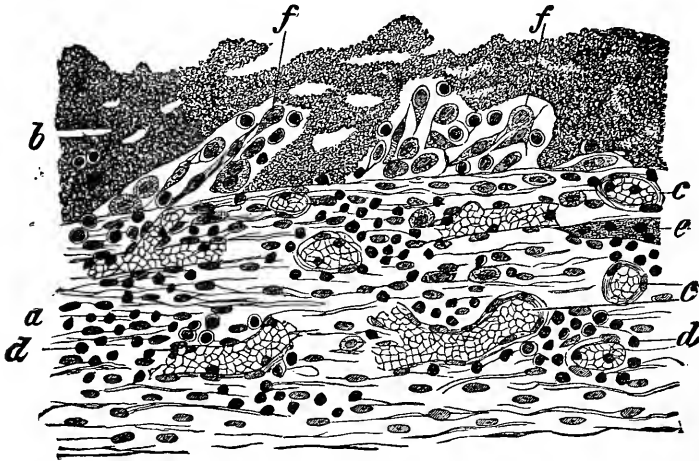


FIG. 26.—Acute pericarditis, sixth day. *a*, epicardium; *b*, fibrin; *c*, dilated distended blood-vessels; *d*, round cells infiltrating the tissues; *e*, lymph-vessels filled with cells and coagula; *f*, fibroblasts in the exudation. $\times 150$. (Ziegler.)

stricture of the auriculo-ventricular ring; or of a combination of two or of all of these forms of morbid change.

4. PERICARDITIS.—Acute inflammation of the pericardium involves the visceral and parietal layers, either universally or locally, and it usually leads to effusion into the sac. As a result commonly of some acute specific process, such as rheumatism or septicæmia, but possibly of toxæmia (Bright's disease) or local injury, the serous surface becomes dull, swollen, and hyperæmic from ordinary inflammatory changes, fibrin forms rapidly upon it, and this comes to be disposed in a peculiarly matted, reticulated, or shaggy fashion in consequence of the continual movements of the heart (*cor villosum*). The intra-pericardial effusion is serous, sero-fibrinous, or hæmorrhagic, and of very variable amount; it usually disappears within a short time, but may remain for an indefinite period.

Sometimes the exudation is purulent or sero-purulent; and whilst this form of pericarditis commonly then proves fatal, it may end in inspissation and calcification of the products and firm adhesion. Examined microscopically, the pericardium is found to be covered with an irregular layer of fibrin, its vessels congested, its proper tissue infiltrated with leucocytes. Presently the fibrin is penetrated from beneath by leucocytes, vascular sprouts, and fibro-blasts, from which a vascularised connective tissue comes to be developed, whilst the fibrin disappears (Fig. 26). The micro-organisms found in connection with pericarditis include streptococci, staphylococci, gonococci, pneumococci, tubercle bacilli, and the bacillus coli communis. Complete reabsorption of the inflammatory products may take place, or opaque patches remain, or adhesions form, whether local or general, by the growth of young connective tissue and vessels between the layers, and in this way the cavity is sometimes completely obliterated—adherent pericardium.

Adherent pericardium.—This condition commonly leads to general enlargement (hypertrophy and dilatation) of the heart; exceptionally to atrophy. *Chronic pericarditis* is another name for adherent pericardium, or for persistent inflammatory effusion in the sac, which is usually tuberculous.

White patches on the visceral pericardium, “milk spots,” are not uncommon on the anterior surface of the right ventricle or near the apex. They consist of small local opacities of the serous covering of the heart, apparently the result of friction against the præcordial parietes and attendant plastic inflammation.

5. PYO-PNEUMO-PERICARDITIS is a rare pathological condition in which the pericardial sac contains pus and foul gas. It is usually the result of the irruption of infective matter from the œsophagus, abdomen, or lungs, or of a wound of the præcordia.

IV. STRUCTURAL CHANGES REFERABLE TO DYNAMIC DISTURBANCES OF THE CIRCULATION

The heart undergoes a variety of changes in its size, substance, and cavities, consequent on disturbances of the movement and pressure of the blood within it and in other parts of the circulatory system.

1. HYPERTROPHY.—This, the most common of all the pathological changes which the heart presents *post mortem*, involves the muscular tissue of the walls of one or more of the cardiac chambers. The individual fibres are increased in size and possibly in number,

and the result is thickening of the walls as determined by section, and of the trabeculæ and papillary muscles, which look coarse and thick, the tissue at the same time being of a deep red colour and firm consistence. The degree of hypertrophy is estimated by measurement of the thickness of the walls; it may amount to one and a half or two inches in the left ventricle, whilst the weight of the organ as a whole in general hypertrophy may be doubled, trebled, or even more. In addition to these characters, hypertrophy of the *left ventricle* gives rise to unnatural elongation of the apex of the heart; and the interventricular septum is thickened and bulges into the right ventricle, which appears relatively very small, particularly in transverse section (Fig. 27). Hypertrophy of the *right*

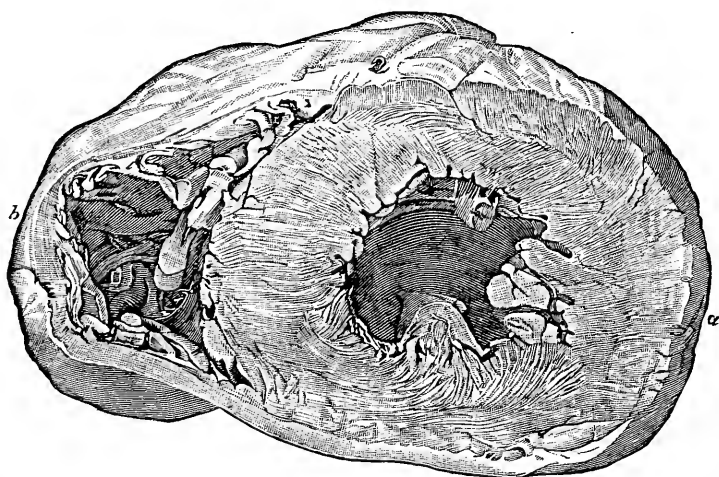


FIG. 27.—Hypertrophy of the left ventricle, produced by insufficiency and stenosis of the aortic valves: transverse section $\frac{3}{4}$. *a*, left ventricle; *b*, right ventricle. (Zeigler.)

ventricle blunts the apex of the heart; increases the thickness of its wall from one-eighth of an inch to half an inch or even more; its tissue is usually peculiarly tough as compared with that of the left ventricle; and the columnæ carneæ stand out prominently as stout, thick, fleshy masses. Pure hypertrophy of the *auricles* is difficult to estimate, and indeed perhaps does not exist.

2. DILATATION.—This term is applied to a state of increased capacity of a chamber or chambers, and every degree of it is met with. Simple dilatation is necessarily developed by thinning of the walls, but in the great majority of cases the condition is associated with hypertrophy, which maintains or augments their proper bulk, and increases the weight of the heart to two or three times the normal. Dilatation also changes the shape of a cardiac cavity,

which becomes saccular, and the ventricles become relatively wider and somewhat rounder in outline.

Enlargement of the heart, whether locally or generally (apart from irregular increase of size due to new growths), is the result of hypertrophy, of dilatation, or of combined hypertrophy and dilatation. Of these three changes, the last-named is the one most commonly present in cardiac enlargement. In some instances hypertrophy accompanies or follows closely upon dilatation; in other instances, dilatation is sequential to hypertrophy, as described at p. 385.

3. THROMBOSIS OF THE CARDIAC CAVITIES.—Two distinct kinds of thrombi are found within the heart. The first, and more familiar, form in the track of the principal blood-currents, particularly in the auricles, at the arterial orifices and in their trunks, and in the meshes of the chordæ. These are variously coloured or decolorised homogeneous clots, deposited before, at, or after death, with very loose or no attachment to the walls. The second kind are often overlooked because they occupy the remote and obscure corners of the heart where the current is weak, including the auricular appendages and the crevices between the trabeculæ of the ventricles. They appear as fleshy polypi, stratified and centrally softened, with considerable adhesion to the walls; occasionally they are free globes. Such thrombi may lead to embolism. The reader will remember that thrombosis is also an element of endocarditis, whether of the valves, or of the parietes consequent on myocardial infarction. The pathology of thrombosis is described in Vol. II. p. 315, in connection with morbid conditions of the blood.

4. STRAINS AND RUPTURES.—The pathological effects of sudden and severe stress falling on the heart are rupture and stretching of the valves, and rupture and stretching of the parietes.

Rupture of the valves has been found both at the arterial and the auriculo-ventricular orifices, but it is a rare lesion; most often it involves structures already unsound. The segments or the chordæ are torn; the ragged borders and ends injure the parts around, setting up vegetative endocarditis; dilatation and hypertrophy follow incompetence and obstruction of the valves, and embolism may occur. Occasionally the lesion is instantly fatal. Stretching of every degree may be traced in valves softened by endocarditis or degeneration.

Rupture of the muscular walls of the heart appears always to be a secondary lesion, the part that gives way—usually the left ventricle—having been weakened by fatty or other degeneration (Fig. 28).

The muscular fibres may also be torn by direct blows on the præcordia, by coronary hæmorrhages, or by bursting of an abscess or cyst. Rupture of the heart is followed by intra-pericardial hæmorrhage — *hæmo-pericardium*—and death.

Stretching of the parietes, when it involves an entire chamber, constitutes dilatation; when it is local, consequent on degeneration, it is known as cardiac aneurysm, which in its turn may rupture.

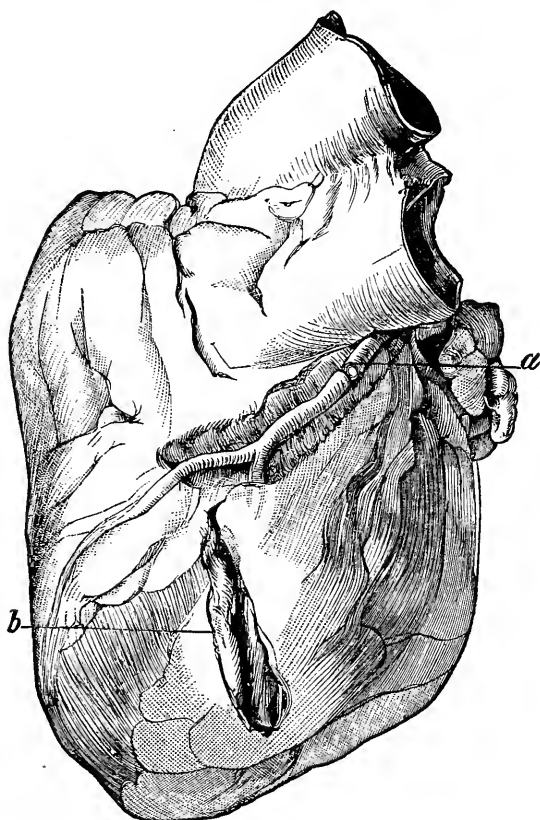


FIG. 28.—Sclerosis of a coronary artery; myomalacia; rupture of the heart. *a*, branch of the left coronary artery occluded by sclerosis and thrombosis; *b*, rupture. (Ziegler.)

V. TRAUMATA

Injuries of the walls and valves of the heart include ruptures and strains, just described, as the effects of violence, wounds, and damage caused by the incursion of foreign bodies, parasites, and emboli. Incised, contused, and gunshot wounds are of

every degree of severity, and of great variety of situation. Hydatids, venous and pulmonary thrombi, air or fat, may burst or be swept into the cardiac chambers, and produce embolism of these or of the arterial orifices.

VI. NEW GROWTHS AND PARASITES

New growths are but seldom met with in the heart. They include carcinoma, sarcoma, lymphoma, myxoma, fibroma, and rarer kinds, carcinomata being secondary formations. Infection occurs sometimes by continuity from the œsophagus, stomach, mediastinum, or lung; sometimes through the blood. The

tumours present themselves on the pericardial or endocardial surface, or they are discovered by section. Occasionally they disturb the relations of the valves; internally they may be covered with thrombi; sometimes they soften.

Tubercle affects the endocardium, myocardium, and pericardium, but except in the last of these situations is rare and of little importance. Tuberculous pericarditis is usually a chronic process characterised by sero-fibrinous effusion, occasionally hæmorrhagic, by a shaggy or partially adherent state of the surfaces of the sac, and by the presence of nodules and caseous masses of the usual appearances.

Syphilis of the coronary vessels and its effects on the myocardium and valves have been already referred to. Gummata of the myocardium present the familiar characters of these lesions according to their age, size, and situation; they are comparatively rare. Occasionally they lead to cardiac aneurysm.

Actinomycosis has been met with in the heart.

Parasites are amongst the rarest of the diseases of the heart, and include echinococcus and cysticercus. They may burst into a cardiac chamber and cause fatal embolism, as may also echinococci that reach the same situation from another organ.

VII. MALFORMATIONS AND CONGENITAL AFFECTIONS OF THE HEART AND PERICARDIUM

The complex process of development of the heart and great arterial trunks is sometimes interrupted or perverted. The result is a variety of congenital abnormalities of the valvular openings and apparatus, of the septa and the several chambers, of the pulmonary artery, aorta, and ductus arteriosus, as well as of the pericardium and even the præcordial parietes. On the one hand, certain obscure influences, acting unfavourably either upon the germ-cell before fecundation, or upon the embryonic elements before the appearance of the individual structures, disturb development directly, and lead to *malformations*. On the other hand, pathogenetic influences that reach the foetal heart from the maternal blood, including the germs or toxines of the acute specific fevers, cause certain *congenital diseases*, mainly inflammatory in their nature, which occur at the mouths of the great vessels and the auriculo-ventricular openings, after the appearance of the individual parts of the heart and the establishment of circulation through it. Whichever their mode of origin, these primary ostial and valvular abnormalities may lead to

secondary defects in the heart of equal importance. Obstruction at the mouths of the pulmonary artery and aorta so disturbs the blood pressure on the two sides of the heart as to interrupt the course of the normal closure of the inter-ventricular and inter-auricular septa, which remain more or less defective or patent, with admixture of the arterial and the venous blood. Further, it is obvious that in some of these abnormalities the systemic circulation through the aorta, and the pulmonic circulation through the pulmonary artery, respectively, can only be effected by patency of the ductus arteriosus, which may thus come to be a necessary accompaniment of foetal lesions of the arterial trunks and ostia, and of septal defects.

ABNORMALITIES AT THE PULMONIC ORIFICE.—These are the most common and most important of congenital affections of the heart, particularly when taken in association with the next class (septal defects) and patent ductus arteriosus. Either the conus arteriosus, the first part of the trunk of the pulmonary artery or the last part of the same, or the pulmonic orifice itself, may be narrowed, constituting pulmonic stenosis. The vessel may then be greatly reduced in size compared with the aorta, and its lumen correspondingly small; complete closure is rare; occasionally the vessel appears to be wanting. The valvular segments may be imperfectly developed, and represented by small buds, or they are united at their edges to form a funnel, and thus they are incompetent. The right auricle and ventricle are enlarged relatively to the left chambers. Slight and unimportant anomalies of the pulmonic cusps, only, also occur.

ABNORMALITIES OF THE SEPTA.—Entire absence of the inter-ventricular muscular septum, which grows from below upwards, is a rare abnormality; defect of its upper portion only is more common; defect of the *pars membranacea* is relatively very common. Complete absence of the inter-auricular septum, which grows from above downwards, is very seldom met with; but simple defects of it are common, and the most familiar of all forms of interrupted cardiac development is patent foramen ovale. It will be understood that in considerable septal deficiencies the cardiac chambers are reduced to three, or even two, in number.

Incomplete, that is, interrupted, closure of the cardiac septa is essentially associated with affections of the great arterial trunks and orifices, and the consequent disturbance of pressure on the two sides of the heart; and in the same way they may be complicated with patent ductus arteriosus.

AFFECTIONS OF THE AORTIC ORIFICE.—These are neither common nor important. The orifice or the conus may be stenosed or even completely impervious, and the cusps imperfectly developed; therewith there are associated other malformations, particularly persistence of the ductus arteriosus and foramen ovale, and the left ventricle is enlarged. The aortic cusps sometimes are anomalous in number, form, and size.

Transposition of the aortic and pulmonic trunks at their origin from the ventricles, respectively, is less often found. As a rule it is associated with the abnormalities of the great arterial trunks just described, with septal defects, and occasionally with misplacements of the heart. The pulmonary artery and aorta sometimes have a common origin from the right ventricle—an uncommon condition which is usually associated with malformations of the trunks.

PATENT DUCTUS ARTERIOSUS.—This is a comparatively common and an important abnormality. As just stated, it is often found along with other congenital affections of the heart and great vascular trunks, particularly obstructive lesions of the pulmonary artery and aorta, and septal defects, of which it may be a necessary accompaniment.

ANOMALIES OF THE AURICULO-VENTRICULAR OPENINGS AND VALVULAR APPARATUS.—These comprise a number of comparatively rare deformities, including stenosis and even complete occlusion of the ostia. Consecutively to these obstructive lesions the foramen ovale and the ductus arteriosus, or the interventricular septum, almost necessarily remains patent.

ABNORMAL DISPOSITION OF THE MUSCULAR BUNDLES of the myocardium and associated fibrous structures is met with in various forms, such as columnæ carneæ in unusual situations, bands or fibrous cords crossing the chambers, local thinning with bulging of the walls (congenital aneurysm of the heart), and others.

CONGENITAL MISPLACEMENTS OF THE HEART.—In *dextro-cardia* the heart lies in the right side of the chest. With it there may or may not be associated transposition of the liver and stomach. *Ectopia cordis* is a form of cardiac displacement in which the heart either lies exposed immediately under the integuments, from defect in the lower part of the sternum and pericardium, or is dislocated downwards through a defective diaphragm.

DEFECTS OF THE PERICARDIUM.—The pericardial sac may be more or less incomplete, or it may be entirely wanting. Malformations of this class are sometimes found along with *ectopia cordis*.

COARCTATION OF THE AORTA.—This abnormality, which occa-

sionally proves to be one of much clinical as well as pathological interest, is situated at the *isthmus aortae*, between the origin of the left subclavian artery and the junction of the ductus arteriosus with the arch. The coarctation or stenosis may be of any degree; or the lumen may be completely obliterated, in which case the ascending arch supplies the vessels of the head and upper limbs, whilst the blood in the descending aorta reaches it solely from the trunk of the pulmonary artery through the ductus arteriosus. In coarctation, short of obliteration, the ascending and transverse portions of the arch are dilated; the aortic valves may be secondarily diseased; and many large anastomotic branches are developed between the subclavian arteries and superior intercostals above, and the intercostals and numerous other branches of the thoracic and abdominal aorta below.

COMBINED CONGENITAL ABNORMALITIES.—Many of the different congenital affections separately described often occur together. Such combinations are variously related to each other. Some may be accidental. Others are essential—for example, rudimentary pulmonary valves in pulmonic stenosis. It has been shown that others are necessarily consecutive, such as patency of the foramen ovale in tricuspid occlusion, and defects of the septa and persistence of the ductus arteriosus in pulmonic obstruction.

Whether originating as malformations or as congenital diseases, cardiac abnormalities disturb the distribution and pressure of the blood within the heart and vascular system in the same manner as do acquired diseases of these organs, and lead to hypertrophy, dilatation, or other structural changes in the different chambers. Further morbid changes occur throughout the circulation, the different viscera become involved, health is impaired, life is shortened, and in many instances extra-uterine existence is altogether impossible.

PHYSICAL EXAMINATION OF THE HEART AND VESSELS

INSPECTION

Inspection consists in an intelligent looking at the præcordia. Being the simplest of all methods of physical examination it is often neglected, and some of the most important observations that could be made are missed. It is remarkable how much can be learned by careful observation of the visible appearances only, both normal and abnormal, but such observations and the inferences drawn from them require to be confirmed by other methods of investigation. It should be carried out deliberately, the light falling obliquely across the front of the patient's chest whatever his posture, so that prominences and pulsations may be easily seen. Many appearances strike the observer's eye bearing on the state of the circulation, such as the vascularity and colour of the integuments, and enlargement of the veins; and his survey naturally extends to the neck, face (including the expression), lips, and ears, as well as to the arms and the upper part of the abdomen; but the present description is limited to the signs immediately connected with the præcordia.

General prominence of the præcordia, corresponding with the left cartilages, the body of the sternum, and the sternal ends of the right cartilages, is common in chronic valvular disease, when it is significant not only of enlargement of the heart (which occurs in all directions, forwards included), but in particular of enlargement at an early age when the chest-walls are less resistant—a point often of great importance. Pericardial effusion may also cause general præcordial bulging. *Local bulgings* of the præcordia are usually aneurysmal. The præcordial interspaces may be prominent in pericarditis with effusion, and the integuments visibly puffy.

The visible præcordial impulse, which is normally present in the fifth left interspace, midway between the vertical nipple line and the left parasternal line, and occupies an area of about a square inch, may be abnormal, in particular the *apex-beat*—that portion of it which lies farthest to the left and downwards. Its situation may be displaced by a great variety of causes. The first of these are (*a*) *intrinsic*, the most important being cardiac enlargement, in which the apex-beat approaches or reaches the left nipple line, or may be found to the left of it, in the fifth, sixth, or seventh interspace, or even lower. In enlargement of the left ventricle the heart is mainly increased in length, and the displacement of the impulse is chiefly downwards. In enlargement of the right ventricle the breadth of the heart is relatively

increased, and the displacement of the impulse is chiefly outwards. (b) The second, *extrinsic*, causes of displacement of the impulse are those of displacement or dislocation of the heart itself, whether outwards, downwards, upwards and outwards, downwards and inwards into the epigastrium, or into the right interspaces. The strength of the visible impulse varies through all degrees; or it may be unequal or irregular. It is often imperceptible. Its area or extent may occupy the whole præcordia or a large section of it; and when so *diffuse*, the impulse is often very complex, comprising visible auricular and ventricular waves of different times. Another occasional sign in connection with the impulse requires particular attention, namely, visible systolic retraction, relapse, or at least dimpling of the softer parts of the præcordia near the apex-beat, an exaggeration of the normal insucking of the parietes, which can be made out in thin persons, and, being due to contraction and emptying of the ventricles, is more marked in enlargement and in pericardial adhesion. Other characters of the impulse and apex-beat are best determined by palpation, under which head the significance of all the different characters of the impulse and apex-beat will be considered.

Besides true cardiac impulse, other pulsations may be visible in the præcordia. Of these the most important are systolic pulsation at the sternal end of the second right interspace in dilatation of the aorta (aortic incompetence, atheroma, aneurysm); visible movements of different parts of the heart and the great vascular trunks in retraction of the anterior borders of the lungs by disease; and aneurysmal pulsations in other parts of the front of the chest.

PALPATION

Palpation, by the application of the hand to the præcordia, is employed mainly to determine the characters of the impulse, particularly the apex-beat, and also palpable retraction of the parietes, diastolic shock, thrills, pericardial friction fremitus and tenderness. Less special signs will be mentioned presently. The palpable signs vary considerably with the patient's posture; when he is erect the impulse can usually be better developed if he lean forward and hold the breath in full expiration. Inspection and palpation are usefully combined, by watching with the eye the movements of the finger placed over the apex-beat. First the hand as a whole is used, then the index finger is pressed into the several interspaces, so that the exact situation and all the characters of the apex-beat may be fully and exactly made out.

Palpable præcordial impulse and apex-beat.—The former of these terms is applied to the total impression of systolic cardiac movement conveyed to the observer's hand, and should be distinguished from the palpable apex-beat, which is a term applied to that portion of the true præcordial impulse which is lowest and farthest to the

left, irrespective of shock or agitation of the chest-wall, which may be conveyed to an indefinite distance beyond the actual limits of the heart. The apex-beat is usually, not invariably, the strongest portion of the impulse. The term is not always carefully used, being often incorrectly applied to the præcordial impulse (quite apart from the apex) when this is found in an abnormal or unexpected situation, *e.g.* to the right of the sternum. Palpation of the impulse confirms and largely supplements the visible signs already described, but it fails to elicit certain characters of the impulse which are visible only. In every instance the observer attempts to determine the situation, strength, quality, and extent of the palpable impulse; and the situation, strength, frequency, extent, and special characters or qualities of the apex-beat. The *situation* of both may be morbidly altered, as described under "Inspection." *Force*, strength, or vigour is much more accurately observed with the hand than with the eye. It varies widely in different diseases and disorders of the heart and under different circumstances. In great cardiac enlargement, after exertion, and in nervous and local inflammatory excitement, the impulse may be so powerful as to forcibly lift and shake the hand of the observer, or his head in auscultation; in cardiac exhaustion or degeneration and in general asthenia, it becomes extremely feeble, and in many instances it is altogether imperceptible. But the student must not fail to observe that the tissues lying between his hand and the heart are elements in determining the force of the impulse: a big, bulky fat chest and emphysema equally reduce the palpable impulse to *nil*, and in many adult or old persons it is represented only by a diffused shock. The force of the beat is often quite unequal or irregular. The *extent of the impulse* is a highly important point, and must always be carefully determined, the hand (as well as the eye) being passed over the area of chest palpably moved and determining its limits. These may be recorded exactly; more commonly, the impulse or the apex-beat is said to be localised, extensive, or diffused, as the case may be. A localised well-defined apex-beat is often significant of pure hypertrophy; a diffused impulse is usually significant of dilatation; widely extensive impulse may be due to excitement only. By *quality of the impulse* or apex-beat is meant special additional characters. These include sudden, sharp, short, thumping, snapping, wobbling, slow, dragging, heavy-shock, and other palpable impressions, which are met with in different diseases and temporary disturbances of the heart. The systolic retraction of the parietes near (usually within) the true apex-beat, visible as described above, is also palpable, and is necessarily followed by diastolic rebound. The different characters of the impulse just described are variously combined, and special names are commonly employed to denote familiar types. A powerful, well-localised, fully developed impulse is said to be *thrusting*, as in hypertrophy of the left ventricle in chronic interstitial nephritis; a powerful, sustained, and

diffused impulse is termed *heaving*, for example the dilated and hypertrophied heart of aortic incompetence. It is necessary to distinguish these special varieties of forcible impulse, characteristic as they are of hypertrophy of the heart, by their thrusting and sustained quality, something more than the violent action which a normal heart temporarily exhibits on exertion. The words *indeterminate*, *indefinite*, and *imperceptible* convey obvious meanings. It may be repeated in this connection that the student has a disposition to disregard, amongst all other negative signs, weakness or absence of the præcordial impulse. An indeterminate type of palpable cardiac action ought at once to arrest his attention, for it might be a phenomenon of radical importance.

Palpable diastolic shock, following abruptly on the impulse, and synchronous with the second sound, normally very weak, may be very distinct in thin individuals and in high systemic or pulmonic tension. It is strikingly developed in aortic aneurysm over the pulsating area, and to a less degree in the second right intercostal space near the sternum when the anterior lung-border is retracted. Palpable diastolic relapse is frequently an element of what is briefly called "the præcordial impulse."

Præcordial thrill.—This morbid sign is best determined by laying the palm of the hand not too heavily on the præcordia, and consists in a palpable sensation of vibrations, jarring or purring. In intensity thrills vary extremely; in time they are either systolic, diastolic, or pre-systolic; in situation they are usually either basic or apical, but may be felt at any part of the præcordia. They are produced by eddies set up in the chambers of the heart or in the great vessels beyond, determined by some obstruction to the blood flow, whether this be by narrowed or roughened ostia, or by impact with an opposing column of blood, as in mitral regurgitation. For their production a certain driving-force is requisite, and, should this fail, the thrill may cease to be appreciated, returning as the muscular power is regained. They usually correspond with loud murmurs, and may be influenced by posture.

Pericardial friction fremitus, systolic and diastolic, formed by the rubbing of the opposed surfaces of the sac in pericarditis, is not a very common sign.

Amongst the palpable signs occasionally met with over the heart, but in no respect peculiar to this situation, are fulness referable to bulging or local tumour, pitting of œdema, and different degrees of resistance to careful pressure. Various sensations of softness, rippling, etc., may be felt over aneurysms occupying the front of the chest; and pericardial effusion may occasionally yield fluctuation.

Referred tenderness.—The front and back of the neck and chest, corresponding with the third and fourth cervical and nine upper dorsal segments, is tested for superficial tenderness, by pinching the skin and subcutaneous tissues between the finger and thumb, or by using the blunt end of a pin. Areas or spots of disturbed sensibility

thus discovered may be recorded diagrammatically. Pure mitral incompetence or mitral stenosis, respectively, is unattended with referred pain; but double mitral lesions are characterised by reflected pain and superficial tenderness over one or more of the fifth, sixth, seventh, eighth, and possibly ninth dorsal areas. Superficial tenderness over the second, third, fourth, and fifth dorsal segments is significant of disease of the aortic valves. At the same time the associated areas of the scalp may be found to be tender; in mitral disease the temporal and fronto-temporal area; in aortic disease the mid-orbital (Figs. 12-15, p. 120).

PERCUSSION

Percussion is employed to determine the præcordial dulness, which is valuable evidence of the size, outline, situation, and relations of the heart, as well as those of the roots of the great vessels and the pericardium. The *superficial* præcordial dulness, corresponding with the portion of the heart left uncovered by the lungs, is easily determined. It is an irregularly triangular area bounded internally by the mid-sternal line from the level of the fourth to that of the sixth costal cartilage; thence, inferiorly, by a line along the sixth left cartilage (where the cardiac is continuous with the hepatic dulness); and externally by an oblique line which passes from the upper extremity of the internal boundary along the fifth left costal cartilage, outwards and downwards, to a point just within the left vertical nipple line, whence it curves inwards to join the inferior limit (see Plate I.). The *deep* præcordial dulness, corresponding with the outline of the entire heart, is more difficult to make out. Two different methods of percussion are employed for this purpose: the one is heavy or firm percussion, which brings out the percussion-sound of the heart through, and at the expense of, the resonance of the lungs overlying it; the other is extremely light or soft percussion, which elicits delicate shades or degrees of pulmonary resonance. The latter is to be preferred, and the student should educate his hands and ears in this method. In the same way the slightest differences in resistance can be readily appreciated with the fingers. Some plan or order of percussing out the area of dulness should be followed, and it is well to percuss along recognised lines. The apex-beat having been located and marked with a pencil, the *transverse* dulness is determined by first percussing from the sternal end of the sixth left costal cartilage towards the apex; and secondly, across the sternum horizontally towards the right; this is then laid down by drawing a straight line connecting the two limits of dulness. The *vertical* dulness is next made out by beginning at the same point and percussing upwards along the left border of the sternum until the full resonance is reached (normally about the third cartilage), and this line is laid down in pencil. Returning again to the

tip of the sixth left costal cartilage, the observer now percusses directly towards the left nipple, and marks the limit of dulness in this direction; by drawing a slightly curved line from the limit of the vertical dulness through this point to the left extremity of the transverse dulness, he delimits the left border of the præcordial dulness and of the heart. Light percussion over the lower end of the sternum and the lower right costal cartilages from the right extremity of the transverse dulness upwards will enable him to delimit the right border of dulness and of the heart in a similar way. In every case percussion should not stop here, but include the delimitation of the liver and lower borders of the lungs, also of the stomach—which is done with the patient recumbent.

Increase of the præcordial dulness towards the left is a sign of enlargement of the heart in this direction—of the left ventricle, of the right ventricle (which in its enlargement carries the left ventricle before it), or of both; also of pericardial effusion. Increase of the transverse dulness towards the right, *i.e.* over the right cartilages within and below the nipple, is significant of enlargement of the right side of the heart, also of pericardial effusion; enlargement of the left ventricle does not increase the transverse dulness towards the right. Moderate increase of the vertical dulness, the sternal end of the third left interspace being absolutely dull, commonly accompanies the transverse increase of cardiac enlargement, and so does increase of dulness along the left border of the heart. When the vertical dulness rises to the second cartilage, or even higher, the pericardial sac is involved, either exudation or its effects occupying the intra-pericardial space around the great vascular trunks.

Diminution of the præcordial dulness, transversely, vertically, or in both directions, accompanies atrophy of the heart. Far more commonly, however, it is significant of pulmonary emphysema, the lungs encroaching on the heart. Similarly, increase of the superficial præcordial dulness, with irregularities of outline, results from pulmonary collapse and from retraction of the lung-borders, particularly in pulmonary tuberculosis and fibrosis. If fibrosis and emphysema occur together, they may neutralise each other in their effects on the præcordial dulness, which then appears to be normal.

Extension of præcordial dulness upwards along the sternum, or the development of a new or independent area of dulness in relation with the manubrium and two upper cartilages, is a valuable sign of dilatation or of actual aneurysm of the aorta, as well as of mediastinal tumours.

AUSCULTATION

Auscultation of the heart is employed to determine the presence and characters of the first and second cardiac sounds and of certain abnormal sounds which are produced by disease within or without the organ.

1. *Changes in the normal sounds.*—The first and second cardiac sounds (p. 301) are altered by disease in respect of their loudness, pitch, quality, rhythm, and relative accentuation.

The *first sound* is increased in loudness by excitement, muscular exertion, hæmorrhage, and certain drugs, such as alcohol, ether, digitalis, and strychnine; therewith it acquires a peculiarly sharp, short, or thumping character. Naturally it is louder in persons with thin chest-walls. The first sound is weak in myocardial disease, particularly the granular degeneration of fevers and fatty degeneration. Its muscular element is lost in syncope. But weakness of the first sound has not always a grave significance. It may be due to emphysema, to thickness of the thoracic parietes, to effusion into the pericardium, or to the presence of a solid mass in the mediastinum. Sometimes it is quite inaudible at the base in perfectly healthy individuals, and indeed may be so in hypertrophy of the left ventricle. A first sound that is dull, or rather toneless and sustained, instead of being loud, clear, and abrupt, is significant of muscular hypertrophy. Thus simple loudness, and still less clearness of the first sound, is no evidence of myocardial vigour, just as its right ventricular element is usually louder than the left ventricular element, and of a short, sharp, snapping quality. The first sound may be *reduplicated*, divided, or tripping, in debility, and sometimes quite independently of disease. When reduplication is followed by an accentuated second sound, a cantering rhythm results (*bruit de galop*), which is met with in chronic Bright's disease and is believed to be significant of a left ventricle labouring against high aortic pressure. Various modifications of the first sound, whether at apex or at base, may be difficult to differentiate from weak murmurs, to which they occasionally are transitions.

The *second sound*, a valuable index of aortic and pulmonic tension (p. 301), may be absolutely louder or weaker as a result of disease that increases or lowers respectively the tension in the greater or lesser circulations. It is much louder and clearer in nervous excitement. Of more clinical value is relative loudness (*accentuation*) of the second sounds in the aortic and pulmonic areas. Accentuated aortic second sound is typically met with in the high arterial tension of Bright's disease; accentuated pulmonic second sound is significant of increased pressure in the pulmonic system consequent on failure of the left ventricle, mitral disease, or chronic pulmonary disease interfering with the circulation in the alveolar walls, such as emphysema and fibrosis. This sign is, however, often masked by the pad of emphysematous lung lying over the base of the heart, or by thickness of the parietes.

Reduplication of the second sound is a much more obvious and a better understood sign than reduplication of the first sound. The second sound, instead of being single or continuous, is divided or discontinuous—that is, consists of two elements which are easily differentiated by the

attentive observer. Besides its occasional occurrence in health (p. 301), reduplication is also heard in high arterial tension, both systemic and pulmonic. It is very marked in mitral stenosis.

The quality of the second sound varies greatly. It may be ringing, as in aortic tension; ringing and hollow, as well as intense, in dilatation and degeneration of the aorta; and it may possess these characters attended with a distinct impact on the solid stethoscope, communicated to the observer's head, in aortic aneurysm (*shock-sound*).

Relative accentuation of the first and second sounds at different points of the præcordia is a feature of importance, and one that is more easily observed by students than most of the other characters of the cardiac sounds. Normally, the first sound is more accentuated than the second over the ventricles, whilst the reverse relation holds over the bases. In chronic Bright's disease the second sound, traced downwards, may be found the more intense at the lower end of the sternum and sixth left cartilage, and thence towards the left apex, where, in some instances, it is still accentuated whilst the first is weak; and it may be heard alone even over the back.

The cardiac sounds are very often *irregular* in loudness, in frequency, and in uniformity of rhythm, as a result of disorder or disease of the heart. Weak, short, "small" first sounds alternate with louder fully developed ones, or occur in unequal groups, followed or separated by a single loud first sound, or perhaps by a systolic murmur. Occasionally the sounds occur in couples, of equal or unequal loudness—*coupled rhythm*. Irregularity of the associated second sound is less appreciable.

Spacing of the cardiac sounds is the name given to disturbances of the rhythm of the two cardiac sounds. When diastole and the diastolic silence are shortened, in acceleration of the heart, particularly in palpitation and tachycardia, the interval of silence between the second sound and the first sound following it, and the interval between the first sound and the second sound, come to be nearly equal, and a "tick-tack" rhythm is developed. On the other hand, the interval between the first and second sounds may be increased so considerably by lengthening of systole (including the systolic silence), in high arterial tension with myocardial weakness, as to equal in length the diastolic silence; the sounds are then again heard equidistant from each other.

ADVENTITIOUS SOUNDS.—The auscultatory signs in connection with diseased conditions of the heart other than the modifications of the normal sounds, above described, are called *murmurs* or *bruits*. Some are formed within the heart—endocardial murmurs or bruits; others within the pericardial sac—pericardial murmurs; others, comparatively rare signs, are formed outside the pericardium (see p. 301).

A. ENDOCARDIAL MURMURS.—Murmurs formed within the chambers of the heart and the two arterial trunks are variously named. According to their time, which should be noted with the finger on the

carotid artery, they are ventricular systolic, ventricular diastolic, and ventricular presystolic—more commonly called systolic, diastolic, and presystolic. According to the area of the præcordia at which they are best or only heard, they are named aortic, pulmonic, mitral, tricuspid. They may be thus summarised: aortic systolic, pulmonic systolic, mitral systolic, and tricuspid systolic; aortic diastolic and pulmonic diastolic; mitral presystolic and tricuspid presystolic. These will now be described; but first must be explained what is meant by the terms aortic, pulmonic, mitral, and tricuspid when they are employed clinically in connection with auscultation.

All the four ostia lying close together, the sounds and murmurs formed in connection with them cannot be differentiated by auscultation practised directly over them. Advantage is taken of the conduction or convection of them by different media, namely, the blood current, the substance of the heart and the chest-walls, to spots or areas on the præcordia where they are severally heard loudest, and thus, as it were, unravelled. A murmur formed during systolic output at or beyond the aortic orifice is naturally carried into the arch, and is best heard at the spot where this lies nearest the surface, namely, the sternal end of the second right intercostal space and cartilage; this spot is accordingly called *the aortic area*. Similarly *the pulmonic area* is a clinical name for the sternal end of the second left intercostal space. A murmur formed in diastole at or near either of these orifices, *i.e.* by escape of blood back into the corresponding ventricle, is best heard over the præcordia at a lower level, not in the aortic or the pulmonic area. A murmur formed before and during systole in connection with the mitral orifice and valves is best heard in the region of the cardiac apex, which is the only part of the heart that directly conveys vibrations of these structures to the chest-walls and the observer's ear; the apical region and about an inch around it in all directions is therefore called *the mitral area*. A murmur formed before or during systole at the tricuspid opening is conducted by the right ventricle to the inferior sternal region and adjoining part of the left mammary region of the chest-wall, where it is best heard; these together constitute clinically *the tricuspid area*. As we shall presently learn, the different murmurs are by no means confined to the areas named, the limits of their convection depending on many circumstances; and for the same reason the limits of the several areas are arbitrary, and have been differently defined by different authorities. It is also necessary to remember that cardiac murmurs, like cardiac sounds, are weakened or deadened by the abnormal intervention of pulmonary tissue, solid masses, or fluid collections, and most frequently of all by abnormally thick parietes of the chest in powerfully built or fat persons.

Endocardial murmurs are phenomena produced either (i.) by pathological changes, or (ii.) by functional disturbances of the structures concerned in the production of the normal cardiac sounds.

(i.) Endocardial murmurs that can be traced to acute or chronic lesions of the ostia and valvular apparatus are called *structural murmurs* or *bruits*. When the lesion interferes with the forward passage of the blood from a full to a relatively empty part of the system immediately beyond, the murmur produced is said to be *obstructive*. When the lesion is such as to permit reflux of blood from a full to a relatively empty chamber behind, a *regurgitant* murmur is the result. Both obstructive and regurgitant murmurs are essentially occasioned by eddies of the moving blood setting in vibration the walls, valves, and associated structures (p. 301); and their characters are influenced by the different factors of the audible phenomena produced within the cardio-vascular system. Thus they vary directly with the force and frequency of the heart. Diminished peripheral resistance (low arterial tension) increases their loudness; high tension has the opposite effect. Partly for the same reason, partly because the blood is less viscous, structural cardiac murmurs are more intense in anæmia. And the posture of the patient, which acts partly by gravity and partly by affecting the rate and vigour of the cardiac contraction, develops or augments certain murmurs, and renders others inaudible or at least more faint, as will be described presently. Thus the loudness of a bruit is no measure of the severity of the lesion.

(ii.) *Functional endocardial murmurs* have their origin in temporary disturbances of the force and frequency of the heart, of the peripheral resistance, and of the state of the blood. Sometimes they are significant of disorder of the auriculo-ventricular ostia and associated valvular apparatus due to myocardial atony, as in chronic debility and acute disease. Or they are phenomena of passing cardiac excitement due to nervous influences, violent muscular efforts, or fever. A fall of pressure in the aortic and pulmonic trunks from diminished peripheral resistance favours the production of functional systolic murmurs in the corresponding areas. *Hæmic* or *anæmic murmurs* originate in the combination of two or more of the preceding causes which usually exist in impoverished conditions of the blood. Functional endocardial and vascular murmurs are always systolic in time.

Aortic systolic murmurs, loudest over the aortic area and manubrium, are audible also at the right sterno-clavicular articulation; under the middle of the right clavicle, and along the right carotid artery; occasionally over the upper part of the back of the chest; and sometimes as low as the mitral area, particularly during cardiac excitement. They are of all degrees of intensity or loudness, occupy a great part of the systolic period, are well sustained to the end, and in quality are either blowing, or rough, or musical. They may be variable in rhythm and loudness. They become louder after exertion, and less loud in rest; but they are sometimes harsher in dorsal cubitus.

Aortic systolic murmurs are signs of morbid conditions of the aortic

opening or cusps, which cause obstruction to the output, and much more commonly of lesions associated with dilatation of the aortic arch. Either form of structural change interferes with the normal systolic wave, and gives rise to eddies in the first part of the aorta. Aortic systolic murmurs are also, but less appropriately, called *aortic obstructive murmurs* and *aortic stenotic murmurs*. The morbid conditions of which they are significant are usually degenerative; they are less frequently due to acute endocarditis and its results; and rarely traumatic. Aortic systolic murmurs are also produced by anæmia.

Pulmonic systolic murmurs, loudest in the pulmonic area, are also audible at the left sterno-clavicular articulation, and immediately under the middle of the left clavicle; they are not conducted into the neck. Unless significant of disease at the pulmonic orifice (the rarest of all acquired valvular lesions), they are, with very few exceptions, hæmic in origin (p. 302). But although thus hæmic in origin, they are by no means invariably of the soft blowing quality which might be expected. Hæmic pulmonic murmur is often accompanied by hæmic murmur at the other orifices and in the arteries, as well as by venous hum in the neck.

Mitral systolic murmurs, the most common of all structural bruits, are best, and sometimes only, heard in the mitral area. They are also audible in the left subaxilla and at the left scapular apex (posterior aspect of the left auricle); occasionally they cannot be traced continuously from front to back; in certain cases they are audible at the right scapular apex, and even over the entire back of the chest. Forwards and upwards they are conveyed very variable distances—sometimes as far as the sternum and even the aortic area; they may be well heard along the lines corresponding with the left and the right borders of the ventricles. In other words, their loudness varies in different instances and under different circumstances. They occupy more of the systolic period than the first sound (which is often easily differentiated); that is, part or all of the first period of silence, often running up to the second sound, well sustained to the end. In quality they are commonly blowing—"bellows murmur"; occasionally rough or musical. They are definitely influenced by posture, sometimes being audible only in dorsal recumbency, a very important consideration. Irregularity, that is, want of uniformity in rhythm, loudness, and distinctness, is a common character of mitral systolic murmurs.

Mitral systolic murmurs are signs of morbid conditions of the mitral opening and cusps, producing incompetence, *i.e.* leakage or regurgitation of blood from the left ventricle to the left auricle during systole. They are accordingly also called *mitral incompetent* and *mitral regurgitant* murmurs. The principal diseases that produce incompetence and these murmurs are endocarditis, chronic inflammatory and degenerative processes, and injury; and besides these, relaxation of the muscular

tissue around the ostium and dilatation of the left ventricle, including the mitral ostium, with or without anæmia.

Tricuspid systolic murmurs are loudest over the tricuspid area, but are sometimes conducted far up along the sternum, and also along the left cartilages into the mitral area. They are usually weak, blowing, soft, and ill-sustained, may be modified by posture, and, above all, are usually temporary, being developed along with symptoms and other signs of failing heart, and disappearing as compensation is restored. Tricuspid systolic murmurs are signs of incompetence of the tricuspid valves, and they are also known as *tricuspid incompetent* and *tricuspid regurgitant* murmurs. Incompetence at this ostium is rarely produced by lesion, but is the result (temporary and variable as the murmur shows) of its dilatation from overstretching of the walls (safety-valve action).

Aortic diastolic murmurs may be heard over the base of the heart and the præcordia corresponding to the ventricles; and in some instances they are conducted more extensively in various directions, into the vessels of the neck, and over the back of the chest. The point of maximum intensity in different instances is the lower extremity of the sternum, the region of the fourth, fifth, and sixth left cartilages, the mitral apex, the sternum at the level of the third cartilages, or the aortic area. Thus, the area of these murmurs, that is, their general loudness, is variable. Commencing abruptly, with or without audible second sound, they are ill sustained, dying away at the end; but they are usually long, occupying a great part of the diastolic period. In quality they are usually blowing, but may be harsh, or musical, or mixed with ringing second sound. They are not sensibly affected by posture.

Aortic diastolic murmurs are significant of structural disease—degenerative, inflammatory, or traumatic—at the aortic orifice and the first part of the aorta, producing valvular incompetence and regurgitation of blood from the aorta into the left ventricle; they are accordingly also named *aortic incompetent* and *aortic regurgitant murmurs*. It is doubtful whether they ever proceed from simple relaxation or functional disturbance of the ostium or valves. When present in aortic aneurysm an aortic diastolic murmur is with few exceptions due to associated aortic valvular disease.

Pulmonic diastolic murmur is very rare. It is loudest over the pulmonic area, and audible over the left cartilages; it may be either soft or harsh in different instances; occasionally it is very weak, localised, and soft.

Pulmonic diastolic murmurs are significant of congenital or acquired disease of the pulmonic orifices and valves, permitting regurgitation; some authorities hold that they may be produced by temporary incompetence from pulmonic over-distension. They are also known as *pulmonic incompetent* and *pulmonic regurgitant murmurs*.

Mitral presystolic murmurs are heard in the mitral area or more extensively, their limits being variable, whilst their maximum intensity is usually just within the apex-beat; they vary much in loudness. Their quality is characteristic, being peculiarly rough or "forcing," like the sound produced by water rushing through a funnel; but it also is variable. Most commonly they occupy the time of auricular systole, and then they are of "gathering" character, growing in intensity until they are abruptly interrupted by a sharp "whacking" first sound. In other instances they occupy the mid-diastolic period, or even follow immediately on the second sound, being difficult or impossible to differentiate from aortic diastolic murmurs; they are then said to be "post-diastolic." Sometimes they are developed by dorsal cubitus; more commonly, when quite inaudible in this posture, they become audible on movement of the patient. They are thus the most inconstant of all cardiac murmurs. These murmurs are signs of disease, inflammatory or of obscure origin, obstructing the entrance of blood from the left auricle into the left ventricle, and are also called *mitral obstructive* and *mitral stenotic* murmurs.

Tricuspid presystolic murmurs are heard in the tricuspid area, and have a similar variety, significance, and nomenclature to those of mitral presystolic murmurs. They are comparatively uncommon.

Combinations of murmurs are frequently met with. Whilst mitral systolic murmur alone is more common than a combination of mitral systolic and presystolic murmurs (*double mitral*), a combination of aortic systolic and diastolic murmurs (*double aortic*) is more common than either singly. The order of frequency of combined murmurs is (1) double mitral murmurs and (2) double aortic murmurs, about equal; (3) double aortic and mitral systolic; (4) aortic diastolic and mitral systolic; (5) all four murmurs; and (6) aortic diastolic and mitral presystolic.

B. PERICARDIAL MURMURS.—Murmurs originating in the pericardium possess a rubbing character, sometimes rough, sometimes soft, sometimes musical like the sound of polishing. They are therefore called pericardial friction murmurs or sounds, or *pericardial friction*. When fully developed they are triple, *i.e.* they possess an auricular systolic, a ventricular systolic, and a ventricular diastolic element, but they are more often double only ("to-and-fro") and occasionally single, whilst their time is seldom strictly coincident with the first and second sounds. Beyond their special frictional quality and time, their characters are best described in contrast with those of endocardial murmurs. Thus, whilst its strength is exceedingly variable, pericardial friction ceases to be audible or becomes strikingly weaker a short distance beyond the limits of the pericardium, although it gives the impression of being easily heard or superficial; it may be limited to an area that bears no relation to the valves or great vessels and that may change from day to day; it is not conducted like murmurs formed at the cardiac orifices; it

becomes louder and harsher on pressure with the stethoscope, and when the breath is held in deep expiration, and either louder or weaker in full inspiration ; it is modified by posture ; and it may be accompanied by the normal cardiac sounds, by one or more endocardial murmurs, or by both. Occasionally it is simulated by pleuritic or mediastinal friction sound.

Pericardial friction is a sign of some morbid process that interferes with the natural smoothness and moistness of the pericardial surfaces. This is practically always pericarditis of some kind ; accompaniments of this, including new growths ; or its effects—such as thickening of the epicardium.

Splashing sounds of very complex rhythm and loud metallic quality are extremely rare physical signs observed in connection with the pericardium. They are produced by the agitation of fluid and gas present together in the sac : pyo-pneumo-pericarditis.

AUSCULTATORY CARDIAC SIGNS FORMED OUTSIDE THE PERICARDIUM.—The most important physical signs of this class include râles of cardiac rhythm, pleuritic friction of cardiac rhythm, various sounds of cardiac rhythm heard in pulmonary cavities, and peculiar metallic sounds of cardiac rhythm audible in pneumo-thorax. These are described in connection with diseases of the lungs and pleura.

THE PULSE

The term “pulse” has come to comprehend, in addition to rhythmical expansion and recoil of the artery, the physical characters of the wall of the vessel itself. These two orders of phenomena are entirely different from each other ; they require to be separately observed in feeling the pulse ; and the significance of each must be individually interpreted.

The pulse is affected by disorders and diseases of the heart and vessels, and conversely it proves to be a most valuable clinical evidence of these, and a guide to prognosis and treatment. The radial artery at the wrist is always examined ; also the brachial, subclavian, carotid, temporal, femoral, and tibials in special cases, whilst the retinal arteries are open to inspection with the ophthalmoscope.

The pulse at the wrist is examined with the fingers, assisted by the eye ; and by means of instruments.

The characters of the pulse to be determined in feeling the pulse comprise the following elements : the size, tension, and state of the arterial wall or coats ; and the frequency, rhythm, amplitude, duration, diastole, force, and time of the pulse-wave. The symmetry of the pulse-wave and wall in the corresponding vessels of the two sides of the body is also noted.

1. *The size* of the pulse is described as being large, medium, small,

or thready ; or it may be unequal, that is, variable or irregular in volume. Size depends on the heart, the vessel-wall, the amount of blood, and the fulness of the venæ comites ; and it varies widely in different morbid conditions of the heart and circulation generally, as well as with the position of the limb. The *paradoxical* pulse is characterised by reversal of the normal (but commonly inappreciable) increase of size during inspiration and diminution during expiration.

2. *The tension or pressure* is estimated by the amount of resistance offered by the artery between the beats to the tip of the finger compressing it gently against the radius. In one case the vessel proves to be incompressible, that is, it feels tense, tight, hard, full, resistant or of high pressure ; in another case it is readily compressed or obliterated by the simple weight of the finger, and feels relaxed, soft, empty, ill-sustained, of low tension, or dicrotic or actually collapsing ; and there are many intermediate degrees of tension, which one attempts to determine and describe as "medium," "moderate," etc. Occasionally the tension is indeterminate. Tension varies widely in different diseases and disorders of the circulation, and in the same case from time to time under different influences, morbid or therapeutical. This is due to the fact that tension is the resultant of at least three variable factors which have to be constantly remembered clinically : cardiac action, peripheral resistance, and volume of blood. At the wrist it is also influenced by the position of the arm in relation to the level of the heart (p. 305).

3. *The condition of the wall* of the radial artery is conveniently investigated after the tension. The observer obliterates the lumen of the vessel by compressing it, and then rolls it under his fingers ; or he traces it up the forearm. It may prove to be variously thickened, and therewith enlarged ; tortuous ; rough from the presence of irregular calcareous particles, or almost uniformly hardened like a pipe-stem. A palpably thickened radial often can be seen ; and tortuosity is best estimated with the eye at the wrist and the elbow and on the temples. A sudden, quick pulse-wave, stretching such an artery as it passes along it, produces an appearance of serpentine movement—the *locomotive* pulse.

4. *The pulse-wave.*—(a) *The frequency or rate* of the pulse is counted with a watch, and in doubtful cases the result is checked by the cardiac sounds, which are much more easily counted than the pulse, particularly when the frequency is great. It is then expressed numerically ; or the indefinite terms "frequent," "rapid," "moderate," or "infrequent" are applied to it. The terms "fast" or "quick," and "slow," also in common use, more properly describe the duration of the pulse-wave, as will be mentioned presently. Frequency is a cardiac element of the pulse, although greatly influenced by the arterial tension and indirectly by the quality of the blood. Physiological disturbances of the rate of the heart are described at p. 296, and the significance of morbid disturbances of rate, including *false infrequency*, at p. 364.

(b) *The rhythm* of the pulse is determined solely by the heart, and disturbances of it are therefore to be studied along with cardiac irregularity (p. 365). It is either regular; or it is irregular, in frequency, or in size, or in force, or in more than one of these elements; or it may be intermittent, or coupled, or otherwise "rhythmically irregular" (p. 366).

(c) *The amplitude* of the pulse-wave depends partly on the force of the heart, partly on the arterial pressure. It is estimated by the amount of excursion of the observer's finger lightly applied to the vessel; by visible pulsation in the arteries of the neck and limbs (for example, in aortic incompetence); and by the altitude or height of the primary wave in the sphygmogram. The more vigorous the left ventricle, with abundant output of blood, and the lower the arterial tension, the greater is the amplitude of the pulse-wave. On the contrary, high arterial tension gives a less ample pulse-wave; and if the ventricular output be small, a very low wave results—the "wiry pulse."

(d) *The duration* of the pulse-wave may be either short or long. *Quick*, *fast*, and *short* are terms correctly applied to a pulse-wave that is

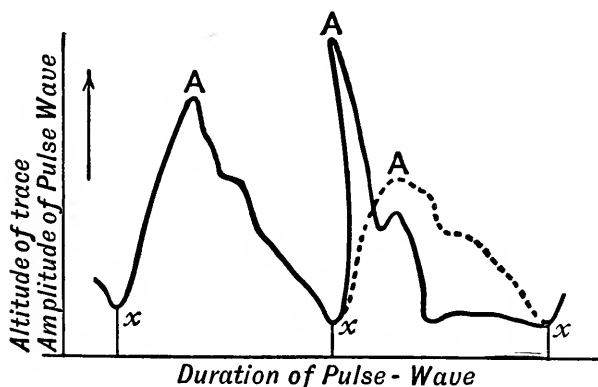


FIG. 29.—Comparative pulse curves to illustrate altitude of the tracing and amplitude of the palpable pulse-wave; duration of the palpable pulse-wave; and relative arterial pressure. *x*, beginning; *A*, crest or summit. The first curve is a normal radial pulse curve (compare Fig. 18, p. 303). The second curve (continuous line) presents greater altitude of the sphygmographic tracing or greater amplitude of the palpable pulse-wave, with short duration of the palpable pulse-wave (quick, fast, or short pulse). The pulse-wave is felt to be sudden, sharp and high, and is as suddenly and quickly lost, so that the final part of the recoil of the vessel is unfelt; the vessel appears to be empty between the beats; and the arterial pressure is low. The third curve (dotted line) presents less altitude of the tracing and less amplitude of the palpable pulse-wave (slow or long pulse). The pulse-wave is felt to be gradual in onset; it rises to a relatively low height; and it persists or slowly falls, so that in the intervals between the beats the arterial pressure is relatively high and the vessel appears to be full.

sudden in its development, and that conveys to the observer an impression of sharpness and of brief duration. Similarly a *slow* or *long* pulse in the proper acceptance of the term is one characterised by a gradually developed, laboured, or even sluggish wave, which appears to persist under the finger and suggests slow emptying of the ventricle. The quick pulse gives a nearly vertical direction to the upstroke in the

primary wave of the sphygmogram; the slow pulse yields an oblique line of ascent.

(e) *Dicrotism* is recognised by the finger as a second wave following on the pulse-wave proper or primary wave, and by full development of the dicrotic wave in the sphygmogram. Its significance is discussed at p. 302. Practically it is accepted as an evidence of low arterial tension.

(f) *The force* of the pulse-wave is estimated by the vigour or vehemence with which it strikes the fingers applied so firmly to the vessel as to obliterate the lumen, or with which it passes underneath the fingers when the compression is gently relaxed. The wave is sometimes strong, sometimes feeble or weak, or it may be imperceptible. This element of the pulse is not wholly cardiac in origin; it depends partly on the size of the vessel and the arterial tension; and the terms "strong pulse" and "weak pulse," as commonly used to express the action of the heart, are often erroneous. Thus a bounding pulse, the resultant of cardiac excitement and low tension from arterial relaxation, occurs in hæmorrhage and fever.

(g) *The time* of the pulse-wave, whether in the radial or in any other artery, in relation to the ventricular systole is estimated with the one hand on the pulse and the other on the apex-beat, or by feeling the pulse during auscultation of the cardiac sounds. The time may be modified by disease in the direction of *delay*, the radial pulse-wave following the apex-beat or the first sound at a distinctly longer interval than normal. This is a common character of the pulse in aortic valvular disease, and in aneurysm and other mediastinal tumours. It is an element referable to the arteries.

5. Both radial pulses should be felt; it is only in this way that *symmetry* can be determined. Asymmetry is necessarily an arterial element of the pulse; it relates to size, tension, and condition of the walls of the artery, and to the force, amplitude, time, and other characters of the wave. It is mainly significant of some local interference with the pulse, whether within or without the lumen. The pulse may be entirely absent at either wrist, or indeed at both wrists.

The *instruments* employed in the clinical investigation of the pulse are the sphygmograph, the sphygmometer, the hæmodynamometer, and the arteriometer. The sphygmograph has been incidentally referred to in the preceding account of the palpable pulse and the characters and interpretation of its tracings. The Hill-Barnard sphygmometer, described at p. 305, and Oliver's hæmodynamometer are used to estimate the arterial pressure. Oliver's arteriometer is designed to measure the calibre of the radial artery.

ARTERIAL SOUNDS AND MURMURS

Audible phenomena connected with the arteries belong to two different orders, namely, conducted sounds and murmurs, and true arterial sounds and murmurs.

1. The cardiac sounds and certain cardiac murmurs are *conducted* into the aorta and its principal branches, and sometimes as far as the radial or even the plantar vessels. The first and second cardiac sounds are easily heard in the carotid and subclavian, the second sound usually the louder of the two. The conduction of aortic systolic murmurs into the cervical vessels, and sometimes far beyond, and the occasional conduction of aortic diastolic murmurs in the same direction, have been already described.

2. *True* arterial sounds or murmurs are formed in the vessels themselves. (a) When an artery is narrowed at one spot by gentle pressure with the finger or the stethoscope, a soft murmur can be heard synchronous with the pulse-wave, *i.e.* with ventricular systole. As the pressure is increased, the murmur becomes more loud and harsh; and under similar conditions it is audible in the smaller arteries. These murmurs can be heard in healthy persons, but are louder in anæmia and cardiac hypertrophy. (b) In some instances of aortic incompetence there is produced in addition to the ventricular-systolic murmur another true arterial murmur, synchronous with *ventricular diastole*, by pressure with the stethoscope on the larger arteries at a distance from the heart, such as the femorals. It is known as Duroziez's murmur or sign. This is a soft murmur, developed by different degrees of pressure in different cases. It is believed to be formed by the backward movement of the blood, which occurs at the point compressed, coincidently with free reflux at the aortic orifice. However, it has also been heard in anæmia and in plumbism.

THE CAPILLARIES

Capillary pulse.—Occasionally the pulse-wave is transmitted through the capillaries, in which it may be seen by employing special methods of examination. If the skin of the forehead be firmly stroked with the finger, the linear flush which is produced will be seen to deepen synchronously with the beats of the radial pulse, and to become paler between them. A similar appearance can be produced in the mucous membrane of the lower lip by everting it, and either lightly compressing it with a glass slide, or stretching it gently; also under the finger-nails by soft pressure on their distal extremity.

Capillary pulsation is significant of low arterial tension, particularly of this in association with aortic incompetence, where the ventricular action is powerful, the output is large, and the relatively empty peripheral vessels with diminished elasticity are suddenly distended by the systolic wave.

THE VEINS

Physical examination of the veins, particularly those of the neck, in respect of the pressure and movements of the blood within them, is

employed in the investigation of morbid conditions of the venous trunks of the thorax and abdomen, as well as of the right chambers of the heart.

1. *Distension* of the jugular veins is best observed in recumbency. If well marked and persistent, as distinguished from passing fulness produced by cough and other forms of forced expiration, distension is one of the signs of obstruction of the corresponding innominate vein or of the superior cava, whether caused by the pressure of tumours (including aneurysm), or by changes in their walls or interference with their lumina. Far more frequently it signifies over-distension and dilatation of the right auricle and ventricle, the result either of over-filling or of failure of these chambers to complete their output in tricuspid disease, certain pulmonary affections, and mitral lesions.

Inspection determines the *condition of the venous valves*, which are prominently marked in distension, unless this is extreme, when they become incompetent. Sudden visible *collapse* of these veins during ventricular diastole is occasionally observed in adherent pericardium.

2. *Visible pulsation* in distended cervical veins is produced by the forces which act upon it *a fronte*, that is, the cardiac movements and the respiratory movements. Distended jugulars then present a double pulsation of cardiac origin—a presystolic or auricular wave, and a systolic or ventricular wave, and these waves are modified rhythmically by inspiration and expiration. Thus venous pulsation as a whole is of a complex, irregular, tremulous character, somewhat difficult to analyse. Jugular pulsation is significant of fulness of the great veins and right chambers of the heart, and usually, but not necessarily, of tricuspid incompetence; indeed, it may be noticed in some healthy individuals when recumbent.

Wave tracings have been obtained from the veins. Venous pulsation in the neck gives a complex tracing, which includes auricular and ventricular waves, the former either physiological or pathological, the latter, when well developed, usually pathological, originating in tricuspid incompetence.

Palpable pulsation in the veins is a very rare physical sign, excepting in connection with certain surgical diseases of these vessels.

3. *Visible reflux current* in the veins of the neck is an important physical sign in certain diseases of the heart. If the external jugular vein be emptied upwards, and the vessel compressed above with the finger, it is found to be filled from below, that is, backwards from the trunk-veins and heart. Venous reflux is usually associated with venous fulness and venous pulsation, already described, but it implies more than these, namely, incompetence of the venous valves in the neck; and, as distinguished from a simple backward wave along distended vessels, it is certain evidence of tricuspid regurgitation.

In connection with the preceding conditions and failure of the heart,

the cervical veins are sometimes palpable as solid cords, a result of thrombosis.

Intimately associated with distension, pulsation, and visible reflux in the cervical veins, and of great clinical importance in connection with cardiac failure, is palpable venous *pulsation of the liver*. It is easily felt with the hand over the right hypochondrium. Systolic in rhythm, it signifies tricuspid regurgitation and reflux through the inferior cava and hepatic veins.

4. *Visible enlargement, fulness, and varicosity of the parietal veins of the thorax and abdomen*, as well as of the upper limbs, are significant of obstruction of one or other of the venous trunks, including the upper and lower cavæ, innominate and subclavians, or of the internal mammary veins. By observing the direction of the current, as before, we can determine whether the distended parietal veins are conveying blood downwards to relieve obstruction within the chest, *e.g.* in thoracic aneurysm; or upwards, as a relief system to a block in the inferior cava or in the portal system.

5. *Venous hum* is the only auscultatory sign of importance connected with the veins. It is usually observed in the neck, and is best heard immediately above the inner end of the right clavicle, between the attachments of the sterno-mastoid muscle, whence it may sometimes be traced over and beyond the manubrium, particularly in children. Its characters are definite and easily recognised. The most important of these is that it is a continuous sound, as distinguished from the interrupted sounds or murmurs connected with the heart and arteries. Whilst continuous it is not uniform, but varies rhythmically with (*a*) inspiration, during which it is louder, and expiration, during which it is weaker; and with (*b*) the cardiac movements, being louder in auricular diastole. The quality of the venous hum is familiarly described as that of the humming-top, whence its French name—*bruit de diable*; sometimes it is fine, sometimes so harsh or coarse as to be roaring; often it is finely whistling; or it breaks on the ear like the “singing” of a kettle beginning to boil. It is influenced by the tension of the cervical muscles and fasciæ, so that the observer can modify it at will, produce it or arrest it, by rotating, extending, or flexing the patient’s head. It is weaker in recumbency. The significance of venous hum is described at p. 301. When loud it is regarded as a sign of anæmia, but a weak hum is frequently heard in perfectly healthy individuals, especially women and children.

A venous hum audible over the manubrium may sometimes be mistaken for an endocardial murmur, particularly a faint diastolic aortic murmur. It must not be confounded with the muscular bruit.

6. *Venous pulse*, that is, a pulse-wave propagated directly through the arterioles and capillaries into the veins, is a rare physical sign, which has been observed on the backs of the hands and feet. True venous

pulse signifies a greatly relaxed condition of the peripheral vessels, possibly in association with aortic incompetence. It may give a tracing with a light lever. It must be distinguished from venous pulsation produced *a fronte*, as in the jugulars, still more rarely seen in these situations.

GENERAL SYMPTOMATOLOGY

The morbid conditions of the heart which have been reviewed in the last section give rise to a variety of disorders of the action and sensibility of the heart, to disturbances of the distribution and pressure of the blood in the different cardiac chambers and the vessels of both the systemic and pulmonic circulations, and to serious interference with the functions of the viscera. The subjective phenomena of these derangements and disabilities constitute the symptoms of heart disease, and are studied with great accuracy as guides to the diagnosis of the pathological conditions which underlie them. Many of these morbid cardiac phenomena prove to be common to a variety of diseases of the heart, being significant or symptomatic simply of circulatory excitement, difficulty, failure, or otherwise; and it is therefore possible to discuss them collectively under the head of general symptomatology, instead of studying them repeatedly in connection with every individual disease in subsequent sections. Further, by pursuing this method of instruction, it is possible to appreciate far more readily the great problems of the pathogeny of affections of the heart and vessels, and the principles on which prognosis and treatment can be rationally practised. The symptoms of cardiac disease, whilst less striking and attractive than its physical signs, are of at least equal importance; and the study of them must on no account be neglected, as often happens, from an exaggerated estimate of the relative value of physical examination.

The general symptomatology of diseases of the heart will now be reviewed under the following heads:—1. Disturbances of the force of the heart; 2. Disturbances of the rate of the heart; 3. Disturbances of both force and rate; 4. Disturbances of the sensibility of the heart; 5. Special disturbances of cardiac action and sensibility.

I. DISTURBANCES OF THE FORCE OF THE HEART

Rhythmic contraction being the automatic function of the cardiac muscle (p. 295), governed by the vagus and sympathetic, disturbances in the force of this contraction will furnish invaluable clinical evidence of the condition and activity of the myocardium

and nervous mechanism of the circulation. To begin with, the signs of vigorous action of the heart, such as are found in perfect health and in cardiac hypertrophy, include a visible and palpable præcordial impulse that is well-defined, localised or extensive, forcible, and thrusting; a first sound that is dull-toned and sustained, particularly in the mitral area, and a second sound that is both loud and clear in the aortic area. The pulse is quiet, forcible, usually regular, and of variable size and tension. All these signs are more fully developed by muscular exercise and by moderate mental excitement.

(a) *Abnormal increase: cardiac excitement.*—The action of the heart is morbidly increased as a result of a variety of the causes and pathological changes studied in the previous sections. Such are severe exertion, or even moderate exertion in certain susceptible subjects (irritable heart); nervous excitement or agitation; functional disorders of the circulation, particularly Graves' disease; some kinds of food and drugs, including coffee, alcohol, ether, chloroform, strychnine, trinitrin, and ammonia; cardiac or pericardial inflammations in the first stage; sthenic fevers; aortic incompetence, with dilatation and hypertrophy of the left ventricle; and sudden interference with or disturbance of systole, as in embolism of the pulmonary artery or rupture of a valve. Morbid increase of the cardiac action also very often occurs paroxysmally in all kinds of cardiac disease and disorder when flatulence, movement, excitement, or sudden rise of pressure in either the systemic or the pulmonic circuit makes excessive demand on the heart. Under these circumstances, the cardiac action is found to be increased, excited, morbidly exaggerated, palpitating, or even tumultuous. The visible and palpable impulse is powerful or violent, and extensive; and the præcordia, and even the parts beyond, are agitated. The first sound is accentuated and less dull, or it may be partly blowing; the second is even more increased in loudness and clearness; or if the sounds be replaced by the murmurs of associated valvular disease, these are also abnormally increased. The pulse is usually more frequent, and may become visible or "throbbing" in the neck; the primary wave is abrupt and stronger, and the tension is usually, but not necessarily, raised. As a rule, the cardiac action becomes perceptible by the patient, who describes it as "palpitation," and complains of præcordial distress; occasionally even the pulse is felt in the ears and throughout the arteries.

(b) *Abnormal feebleness or decrease: Cardiac weakness.*—Morbid feebleness of the rhythmic action of the heart occurs in nervous

and muscular exhaustion ; in some emotional states ; as an effect of certain drugs and poisons, such as chloroform in the third stage, antimony, ipecacuanha, and all irritant and corrosive poisons ; in acute diseases and injuries of the abdomen ; in shock and severe pain from sudden injury of any part, including the heart itself ; and in many diseases of the heart where the nutrition of the myocardium is impaired by degenerative or inflammatory processes, exhausted by chronic strain, or mechanically interfered with in its contractions. Diminished or weak contraction of the heart may be of every degree, even to apparently complete cessation. The visible and palpable impulse is diffuse, feeble, or entirely imperceptible. The first cardiac sound is weak, but sharp, short, and clear ; its feebleness may advance until it disappears. The second sound is less obviously changed in character, and its loudness at the aortic and pulmonic areas differs according to the relative tensions in the two circuits. In many instances the impulse and sounds are entirely arrhythmical—asystolism. Various distressing sensations of præcordial and epigastric oppression, irregular palpitation, faintness, anxiety and possibly pain, pallor, dyspnœa, and arrest of movement, one or more in different associations, accompany these physical signs. The radial pulse is small, its tension cannot be readily estimated, but is probably very low ; the wave is feeble, sometimes frequent, sometimes infrequent ; it often intermits or is irregular, and it may be running.

Increasing feebleness of the cardiac contractions is an ordinary manifestation of progressive disease of the heart. The different phenomena just enumerated make their appearance together, and, unless treatment be employed, they advance and may end in death by failure of the heart. Such advance, as a rule, is not uniform, however. Sometimes a slight return of strength occurs temporarily. Sometimes there may be a brief attack of morbid excitement or palpitation. Sooner or later there comes an acute and possibly sudden manifestation of increase of weakness, a paroxysm of grave and *rapid failure of the heart*, with dyspnœa, præcordial distress, or actual pain in which the patient may die. Distress amounts to agony ; the lips are livid, the skin is cold and covered with sweat ; after brief attempts to obtain relief by constant changes of posture, the patient collapses ; noisy liquid râles (rattles) are heard in the throat ; a leaden pallor overspreads the body ; consciousness fails ; respiration becomes less and less loud ; and the pulse, which has become more and more irregular, may almost suddenly cease. An attack of this severity commonly ends in death ; but recovery is not impossible.

(c) *Syncope*.—Sudden failure of the heart is known as a faint or syncope. The phenomena of the commencement of a faint are familiar. The patient suddenly turns pale, has a sense of sinking in the epigastrium, weakness and giddiness; he sways, and if standing has a tendency to fall. The eyelids tremble, the globes turn upwards, sight is impaired, sounds are heard “far away,” and consciousness rapidly fades. The pulse fails, becoming either frequent, small and feeble, or infrequent, irregular or intermittent. Respiration is weak and irregular. By this time the muscles are relaxed; the patient sinks down, unconscious and apparently inanimate. The skin is pallid, and rapidly becomes cold and damp; the eyes are closed; the pupils are dilated; the action of the heart and the pulse are almost imperceptible; and the breathing is infrequent and sighing. General convulsions sometimes occur. Presently a slight increase is observed of the pulse, with movements of the features and fingers, and deeper sighs; the cardiac action grows stronger; colour slowly returns in the lips and face; and sensibility and intelligence are regained. In other instances, however, no improvement occurs, and syncope ends in death, especially when it is the result of hæmorrhage or the heart is diseased.

Syncope consists in sudden failure of the heart, as the result either of structural disease; of sudden disturbance of the nervous impulses to it, particularly in severe injuries, in emotional depression or in visceral disorder; of impurity or poisoning of the blood in the coronary arteries; or of failure in the return of blood to its chambers from the veins. The last-named cause is very important; it is met with in severe hæmorrhage, and in the deficient filling of the right ventricle which attends loss of muscular tone and determination of blood to the venous side when a patient suddenly assumes the erect posture in convalescence from a prolonged illness.

2. DISTURBANCES OF THE RATE OF THE HEART

Increased frequency or acceleration of the heart is one of the commonest of clinical phenomena. The rate rises from the ordinary, 70 to 90 in the adult, to any number under 200, and occasionally beyond this, and the patient may or may not be sensible of the rapid action. The significance of rise of frequency of the heart is very different in different instances. Irrespective of the many causes of physiological acceleration, such as excitement and

exertion (p. 298), it is, with few exceptions, one of the results of fall of arterial pressure. In this way diminished cardiac force leads indirectly to acceleration, which is compensatory in its effect on the circulation ; and, conversely, a rise in the pulse-rate is employed to estimate the strength of the heart, and of the system as a whole in disease of every kind. Some drugs have the same effect, such as the nitrites. In connection with cardiac disease, increased frequency of the heart most commonly has the same significance. Thus it is met with in cardiac failure, often along with irregularity, both continuously but of moderate degree (100-150), and paroxysmally as a result of movement, excitement, or local oppression. Rarely it constitutes true *tachycardia* or heart-hurry, the rate rising unexpectedly to, say, 160, and falling after hours or days, perhaps suddenly, to exactly one-half, when the heart is relieved, or possibly without obvious cause. In a special but mixed group of cases of circulatory disturbance, a marked increase of the pulse-rate is a cardinal phenomenon. Such are, first and far more commonly, Graves' disease and allied conditions (Vol. II. p. 155) ; and, secondly, a remarkable and still obscure type of cardiac affection, of which rapid heart (110-260 per minute) is the only characteristic feature, and which may end in sudden death. Lastly, tachycardia occurs as a temporary disturbance, whether with or without cardiac disease. It is then suddenly developed, possibly as a result of gastric disorder, lasts for some hours only, and may as suddenly disappear.

Diminished frequency, infrequency, or retardation of the heart is relatively uncommon, whether in cardiac disease or in other morbid conditions. It is usually estimated by feeling the pulse ; but in every instance the result should be checked by the cardiac sounds, so as to exclude the fallacy of *false infrequency*, which depends on the failure of some of the pulse-waves to reach the wrist. In true infrequency, the rate may fall to 50, 30, or 20 ; and even 7 per minute is said to have been observed

The causes of diminished frequency of the cardiac contraction are various (p. 297). It is a familiar effect of certain drugs or poisons, particularly digitalis and its congeners, lead, the biliary acids, and specific toxins ; and it is a physiological result of high arterial pressure from any cause. Amongst cardiac diseases it is mostly significant of acute or chronic myocardial degenerations, with dilatation. In a similar way the cardiac sounds may be observed to become infrequent as well as feeble when a patient turns faint during præcordial auscultation, and they may be both infrequent and faint in debilitated subjects. The other affections in which

the cardiac rate falls are mainly cerebral and spinal affections, lesions of the vagus, and reflex morbid conditions of the abdominal organs. Infrequency is often associated with syncopal and epileptiform seizures. Marked infrequency of the heart, when it is habitual or paroxysmal, is called *bradycardia*.

3. ASSOCIATED DISTURBANCES OF FORCE AND RATE: IRREGULARITY

The force, frequency, and rhythm of the cardiac contractions are often disturbed together. Paroxysmal acceleration is usually associated with increased or even violent præcordial impulse; and feebleness of action may accompany either tachycardia or bradycardia.

Cardiac irregularity is the result of many different morbid conditions. Of these the most important are structural diseases, particularly mitral lesions and degeneration of the myocardium, which respectively appear to interfere with the relative pressures within the left auricle and ventricle, and with the conductivity and chemical stability of the musculature of the heart. Certain poisons, such as tobacco, digitalis, and uric acid (gout); nervous influences, whether central (emotional) or reflex; affections of the vagus and sympathetic trunks in their course; local oppression of the heart, especially by flatulent distension of the stomach or pleural effusions; and disturbances of the respiratory rhythm, are less frequent but important causes of cardiac irregularity. Occasionally it is purely physiological, particularly in children and in certain individuals (just as it is in the dog), and this whether habitually or on slight disturbance of health. Onset of irregularity or intermittence under observation—for example, during general anæsthesia, or in the course of an acute disease like pneumonia or typhoid fever—is evidence of grave myocardial asthenia, and the disappearance of it is correspondingly favourable. Irregularity of the action of the heart is determined by inspection, palpation, and auscultation of the præcordia; by examination of the pulse; and by ascertaining the sensations of the patient. These different methods should be combined in every instance in order to eliminate fallacies. Force and rate of contraction are usually disturbed together, and the rhythm is completely disordered. Contractions of every variety of force follow each other at quite unequal intervals; or a beat is wanting or missed occasionally—*intermittence*; or several short, rapid, weak beats occur in succession, causing a sensation of fluttering to the patient and to the palpating hand—*tremor cordis*. In some

instances there is rhythmical irregularity, weak contractions or intermissions occurring at regular intervals or in groups or series—coupled rhythm, associated with the *pulsus bigeminus*, *pulsus trigeminus*, etc. (see p. 296).

4. DISTURBANCES OF THE SENSIBILITY OF THE HEART

The healthy man is unconscious of the existence, seat, and action of the heart; but any of the influences mentioned in the former sections, such as exertion, nervous excitement, food, stimulants, and drugs, which disturb the force and frequency of its action, may make it perceptible. Thus the heart has popularly come to be associated with activity, courage, and vigour, and to be regarded as the seat of the affections and other emotions, which in part find expression in cardiac and vascular phenomena. In disorder and disease of the heart a variety of abnormal sensations are developed and referred to this organ, or the related parts of the chest; and consciousness of the existence and condition of the heart may be significant of serious disorder or disease.

The simplest of these is “*consciousness of having a heart*”; the heart is simply “felt,” or it may be “uncomfortable.” Next to these in degree are *sensations of movement*, or beating of the heart within the chest. This is the mildest form of palpitation. The patient may be able to describe the beating as regular or irregular, as strong or violent, as fluttering, rolling, tumbling, etc.; or, on the other hand, as feeble, as halting or intermittent, or—most striking of all—as “stopped” or arrested—a distressing or anxious sense of the absence of something normally unfelt! An important set of morbid præcordial sensations appear to be significant of increased intra-cardiac pressure. They are described as *oppression*, weight, fulness, distension, tightness, or as of being grasped, usually post-sternal in situation. With these, or independently, complaint is made of choking—*globus*—referred to the thyroid region. *Faintness* is one of the most important subjective phenomena connected with the circulation. It is of many degrees, and associated with a sense of something rising from the heart to the head, of “passing,” of “things looking far away,” of sounds growing faint, of weakness, and perhaps of impending death. Possibly giddiness and sweating may occur at the same time. There are great varieties of *præcordial pain*, in character, severity, distribution, and associations, such as a continuous ache in the left mammary region, intermittent prickings or dartings through the same part, and sudden severe pain

in the sternal region, passing through to the back, up into the neck, and down into the arms. In its full development præcordial pain usually takes the form of angina pectoris, to be presently described.

In addition to these varieties of præcordial pain of truly cardiac origin, complaint may be made of pain over the heart that arises in other structures. Such are intercostal neuralgia and myalgia and periosteal pains, situated in the parietes; pleuritic and pulmonary pains; gastric distress referred to the lower sternal region; and the left sub-mammary pain often met with in women.

Sensory impressions originating in the heart are carried by the cardiac nerves to the sympathetic ganglia, and thence through the white rami to the posterior-root ganglia of the dorsal nerves, from the second to the eighth inclusive, where they come into relation with the sensory fibres from the pectoral region, upper limb, shoulder, neck, and head. This relation enables us to account for two important features connected with cardiac and præcordial pain. The first of these is its distribution and spread when severe—the fact that cardiac impressions are not referred to the heart but to the distribution of the far more sensitive cutaneous nerves, with which it is related, namely those of the chest-walls and upper limbs, particularly of the left side of the body. The second is the hyper-sensibility of the same parts that accompanies the pain in cardiac disease, or indeed may exist without it. If a painless sensory impulse be produced in the cutaneous nerves of these parts by means of pinching or pressure, it is modified as it passes through the posterior-root ganglia, the activity of which has been disturbed by the sensory impression proceeding from the heart; it becomes exaggerated or otherwise altered; and on reaching the cerebrum it is perceived as a sensation of pain or tenderness in the peripheral segments or spots. The clinical observer has thus two means of determining the condition of cardiac sensibility: first, by observing the distribution of referred pains; second, by searching for cutaneous hyperæsthesia by gently pinching or pressing the segmental areas known to correspond with the heart. These areas are represented in Figs. 12-15 (pp. 120 and 121). It will be seen that the principal areas related to the heart occupy the greater part of the front of the chest and epigastrium, the back of the chest, and the inner aspect of the arm at least as far as the elbow. The left side of the chest and neck, and the left arm, are far more commonly the seat of referred cardiac sensations and of hyperæsthesia than the right, because of the intimate connection of the left ventricle and auricle (the chambers most subjected to strain and affected in

disease) with the sympathetic branches and the sensory centres of the left side.

5. SPECIAL FORMS OF DISTURBED CARDIAC ACTION AND SENSATION

(1) *Palpitation of the heart* is a term applied, first, to rapid and excited or violent cardiac action, as it is observed by means of inspection, palpation, and auscultation ; secondly, to increased and labouring action of the heart as it is felt subjectively by the patient. When fully developed it is appreciable both by observer and patient. The impulse is extensive, as well as powerful and frequent ; the first sound is loud and intense, and may be audible off the chest ; the second is ringing. A temporary systolic murmur may be audible in the mitral area ; or both sounds may present a rubbing character ; and the cervical vessels throb. The beat of the heart is heard by the patient in his ears as well as felt in the chest ; there is some sense of præcordial distress ; but neither pain, dyspnœa, nor faintness is a necessary accompaniment of simple palpitation.

(2) *Præcordial distress* is compounded of different forms of disturbed cardiac sensations and actions—of fulness, oppression, pain, faintness, irregular or halting pulsation or palpitation, of globus, of indescribable alarm, anguish, and dread of impending dissolution, which is also expressed in the anxious countenance and staring eyes ; and therewith there may be associated some form of disordered breathing, and either arrest of bodily movement or the reverse—an anxious restlessness, flatulent eructations, and even vomiting. In association with grave structural disease, præcordial distress is attended with great suffering. The cardiac action is violent, rapid, and usually irregular in time and force. The visible and palpable impulse is widely distributed over the præcordia and epigastrium, and of a thumping or heaving character ; the entire chest is agitated ; the cervical vessels throb powerfully ; the face is anxious and imploring. The sufferer complains of the beating of his heart, of great præcordial distress, and of dyspnœa, which compel him to sit up in bed and lean the head forwards and downwards, or to throw it backwards with a gasp and a look of despair. Often there is an urgent desire to produce eructation, which gives remarkable relief for a time.

(3) *Angina pectoris*.—This is a name given to an attack of acute distress in the chest, which consists of at least three elements, namely, pain, faintness, and a sense of dying. It is not a disease *per se*, but one of the manifestations of different pathological

changes; and the phenomena of an attack are accordingly very various. On this account it is usual to describe several varieties or types of the affection:—

(a) *Severe and fatal form: classical angina pectoris.*—The patient, usually a man past middle age, under the influence of exertion or excitement, or on meeting a cold wind, is suddenly seized with pain in the præcordia, which is severe, violent, or even excruciating, and sharp or neuralgic in character; it passes upwards and backwards into the neck and left scapular region, and down the left arm possibly as far as the fingers. Therewith a sense of faintness overspreads the patient; and he feels that the action of the heart is disturbed or arrested, that the chest is oppressed or grasped, and that he is dying. Voluntary movement is arrested; the sufferer stands still or clings to the nearest support; the breathing is feeble or arrested, with a feeling of want of breath; his countenance is pale, anxious, distressed, or imploring; the lips are pallid or livid; the surface is covered with a cold sweat. The pulse varies: it may be regular or irregular, frequent or infrequent; its tension has been accurately observed to be high in some instances; its force is feeble, and the arterial wall often proves to be degenerated. After a period of a few moments the attack passes off, leaving a sense of relief, but exhaustion and local soreness; or the patient dies in the brief paroxysm. If he recover, the angina returns after an uncertain interval once or more often; ultimately it proves fatal. Occasional phenomena of classical angina are vomiting and violent—possibly involuntary—action of the bowels; and very different descriptions of the præcordial pain and distress are given by different patients. The distribution of the pain is often less extensive; occasionally the right upper limb, instead of the left, or in addition to it, is involved. It is accompanied or followed by superficial tenderness of the corresponding parts.

Angina pectoris of this grave type has been proved to be associated with fatty and fibroid degenerations of the myocardium; valvular disease, particularly aortic incompetence; acute pericarditis and adherent pericardium; diseases of the coronary arteries; aortic degenerations and aneurysm, and arterial sclerosis; and growths and other morbid conditions of the mediastinum and cardiac plexus. Occasionally no coarse lesion has been found.

(b) *Milder form: toxic or diathetic angina pectoris.*—This type is different from classical angina mainly in being less severe and never fatal—unless it pass into the severe type by the supervention of cardio-vascular disease. Attacks recur at short intervals, on

definite and moderate provocation, such as stooping or smoking tobacco, and this for weeks or months on end. Further, a liability to attacks continues for many years. The phenomena of the seizures are more varied and variable, including the degree, the precise character, duration, and distribution of the pain, the disturbance of cardiac action, the faintness and lethal sensations ; whilst discharges of flatulence and sudden evacuation of the stomach and bowels are relatively prominent, and hæmoptysis may occur.

This form occurs in connection with toxic affections of the cardiac and vascular structures, such as gout and chronic tobacco-poisoning ; with luxurious habits, chronic alcoholism, and worrying sedentary occupations, interrupted by irregular hasty meals and spasmodic recourse to severe muscular exertion by way of correction of hygienic errors. It may also be an effect of acute febrile processes, particularly influenza.

(c) *False angina pectoris*.—Pseudo- or spurious angina is a purely nervous, often hysterical, affection. It is almost confined to women, single or married, and is intimately associated with other neurotic manifestations. Its specific features are the great variety of disturbed sensations in the præcordia, besides pain, such as heat, burning, or weight ; the wide radiation of the pain into the neck, head, and even the left leg ; the prominence of globus ; the association of palpitation of the heart and bodily restlessness ; the occurrence and persistence of heavy, useless, cold, numbed sensations, and possibly redness and swelling, of the left arm ; complaint of intense heartlessness in the attack, and relief by weeping, in which the acute paroxysm usually ends after lasting for hours ; the recurrence of such seizures at varying intervals for weeks or months ; and the absence of physical signs of structural disease.

(4) *Cardiac failure*.—The chief clinical phenomena of fully developed failure of the heart are disturbances of cardiac action and sensation, respiratory difficulty, visceral disorders, and dropsy. The patient reclines semi-erect in bed or in a chair, or he moves restlessly from the one to the other ; in advanced cases his head falls forward on his forearms resting on a support. His face is expressive of suffering and anxiety ; it is puffy, dusky, slightly jaundiced, and yet essentially anæmic ; the lips, nose, ears, and hands are livid, cold, and swollen ; the eyes are suffused. Breathing is frequent, exaggerated, especially on movement, or paroxysmally without obvious cause, very distressing, sighing or gasping, and it is broken by frequent groans and short, single hacking coughs, which end in the expectoration of frothy-white or blood-stained sputa. Complaint is made of præcordial

distress, pain, and breathlessness in many forms (p. 373). When the heart is examined, the præcordial impulse is found to be feeble, diffused, frequent, and irregular, both by inspection and palpation; it may be altogether lost; it is often partly epigastric. The area of præcordial dulness is increased in all directions, but mainly transversely both to left and right. This association of signs of enlargement with feebleness and arrhythmia is highly characteristic. The first sound grows small and weak, loses its muscular element, or is reduplicated, especially in the failing heart of Bright's disease; the second sound varies with the primary lesion, and if it be loud from high arterial tension it gives with the weak reduplicated first sound a characteristic auscultatory sign—the cantering rhythm or *bruit de galop*. If valvular murmurs have existed previously, they may continue audible; or they may have become variously modified, weaker, or lost as a result of deficient parietal force; only small, short, frequent, unequal sounds are then heard, or these with an occasional weak murmur—asystolism. In other instances, previous murmurs are accompanied or replaced by new systolic murmurs in the mitral or tricuspid areas, significant of leakage from fresh dilatation of the ostia; these are variable and transient. At the same time, the signs of bronchial catarrh, pulmonary congestion and œdema, or even infarction, with or without pleural effusion, are discovered in the chest. The pulse, as a rule, proves to be smaller, less regular, much more frequent, and less forcible than before; its other characters vary greatly in the different forms of cardiac disease. The veins of the neck are distended, and present visible pulsation, and sometimes true backward current or regurgitation from the right chambers of the heart. Similarly, pulsation may be found in the liver, which is also uniformly enlarged and tender. The legs and lower parts of the trunk are œdematous. Physical examination often reveals effusions into the peritoneal, pleural, and pericardial sacs—one or more. The urine undergoes the important changes characteristic of failure of arterial and rise of venous pressure (p. 373). Appetite and digestion are variously deranged. Both may be preserved sufficiently to enable light solids to be taken and assimilated without discomfort; or flatulence, acidity, and vomiting may occur and seriously aggravate the patient's distress and danger. The bowels may be either constipated, or relaxed from catarrh. Slight jaundice of the integuments and urine testify to the existence of hepatic disorder; and this is confirmed by the discovery of the physical signs of mechanical congestion of the liver already mentioned. The cerebral condition is

usually miserable; sleep is impossible, restless, or obtained in snatches broken by sudden startings and fearful awakenings; or the patient drifts into a state of dulness, apathy, torpor, or semi-coma, which may end in death.

PHENOMENA IN CONNECTION WITH THE MORE CLOSELY
ASSOCIATED ORGANS IN CARDIAC DISEASE

In addition to the phenomena of cardiac disease just described, which are immediately referable to the circulation itself, useful means of clinically investigating the condition of the heart are furnished by disturbances of the organs more intimately associated with it. These are the lungs, liver, alimentary canal, kidneys, and nervous system, and along with these should be taken the blood and the lymphatic system. The phenomena under consideration are chiefly referable to cardiac failure, inadequacy or temporary embarrassment, and mechanical congestion of the viscera, produced by the process of back-working, which spends itself in swellings, hæmorrhages, catarrhs, dropsies, and other disturbances of these parts. With these phenomena of engorgement of the venous side of the circulation, which can be traced to failure of the heart to complete its output or sufficiently empty itself in systole, there is necessarily associated anæmia from insufficient filling of the arterial system, the phenomena of which are valuable clinical evidences of cardiac disease, including the colour of the integuments—particularly of the face—debility, dyspnœa, giddiness, faintness, and noises in the head and ears. Underfilling of the arteries and overfilling of the veins distributed to the extremities of the limbs together give rise to coldness, lividity, or even cyanosis, characteristic of failure of the peripheral circulation, which can be traced to a central cause. Finally, the extensive disturbances of the circulation in the viscera and other parts just referred to, particularly those of the lungs, liver, alimentary canal, and kidneys, gradually produce a kind of toxæmia, partly by imperfect depuration of the blood and partly by absorption of the poisonous products of disordered metabolism. The principal of the phenomena of cardiac disease originating in this indirect way will now be more fully noticed.

Disturbances of respiration.—The most important of these is *dyspnœa*. This is met with of every possible degree of severity—on exertion, at rest, or in bed; it is constant, intermittent, paroxysmal (cardiac asthma), or periodic; ordinary or orthopnoical,

with or without working of the *alæ nasi*; simple, or intimately associated with different kinds of distress in the chest, heart, and other parts. Dyspnœa in connection with diseases of the heart is symptomatic of a long series of different states or effects of cardiac difficulty and circulatory failure, which it automatically relieves (p. 311). The principal of these are: failure of the left side of the heart, of the right side, or of both; and as consequences of these the following morbid conditions of the respiratory organs—mechanical congestion, œdema, and infarction of the lungs; mechanical congestion, œdema, catarrh, and inflammation of the bronchi; secondary pneumonia; and pleural effusions, whether passive or inflammatory. Other causes of dyspnœa referable to cardiac affections are—primary engorgement of the right side of the heart, for example, in violent exertion or pulmonic embolism; interference with the action of the diaphragm, or actual pressure upwards through it upon the heart and lungs, by flatulent distension of the stomach and bowels, and ascites due to portal congestion; general anæmia; cerebral anæmia and asphyxia; and renal impairment. The differentiation of these causes of cardiac dyspnœa from each other is of great importance in treatment.

Cough of cardiac origin is characterised by its short, sharp, single hack; it accompanies dyspnœa, and is usually induced by movement. It signifies irritation of the pulmonary alveoli and bronchi by mechanical congestion momentarily increased. Naturally other forms of cough accompany the different causes of dyspnœa just enumerated. The *sputa* associated with diseases of the heart usually consist of frothy mucus; or this is stained or streaked with blood, or there may be free *hæmoptysis*—all evidences of various degrees of pulmonary congestion and cardiac insufficiency, and possibly of pulmonary infarction.

Disturbances of the kidneys.—The urine furnishes one of the most available, simple, and certain means of estimating the state of the arterial and venous circulation, of the blood pressure, and of the heart; of the progress of any case of cardiac disease; and of the action and value of remedies. The amount of urine passed daily should always be measured and recorded (along with the number and character of the stools), thus furnishing an observed fact that is invaluable because definite and unprejudiced, as compared with the pulse, for example. When the heart fails, the volume of urine falls to 30, 20, 10 fluid ounces, or less. Therewith the secretion rises in specific gravity and acidity, is high-coloured, turbid, and often jaundiced, deposits urates heavily, and proves to contain a relative

excess of solids, bile, hyaline or granular casts, visible urates and free uric acid crystals, albumen, and sometimes blood. Practically speaking, all these abnormal characters increase and decrease in degree, respectively, with increase and decrease of failure of the heart; and if the heart and circulation be restored, the urine regains its normal characters.

Disturbances of the alimentary system.—These are often valuable guides to the state of the heart. On the one hand, epigastric discomfort, flatulence, and vomiting of food, mucus and even traces of blood, are among the symptoms of mechanical congestion of the gastric veins and consequent catarrh, that is, of cardiac failure. On the other hand, the effect of a full or indigestible meal in producing præcordial anxiety, palpitation, and faintness may be evidence of weakness of the walls of the heart, and often the first. Intestinal fluxes and hæmorrhoids confirm the suspicion of portal congestion and cardiac incompetency. The signs of increasing enlargement, tenderness, and possibly pulsation of the liver are amongst the routine means of determining failure of the heart, and their disappearance is a measure of returning force. A slightly jaundiced tint of the face in association with lividity and anxiety is characteristic of heart disease with failure.

Disturbances of the nervous system.—Disease of the heart disturbs both the arterial and the venous circulation within the cranium so much and so readily in consequence of the powerful influence of gravity (see p. 313), that cerebral phenomena are amongst the most striking of cardiac symptoms. Faintness and syncope at once suggest acute failure of the arterial supply to the brain. Other cerebral or associated disturbances are vertigo and tinnitus; headache; simple insomnia, which is often distressing and obstinate; heaviness and somnolence; such emotional disorders as anguish, anxiety, heartlessness, dread of death, of being left alone, of being confined in a close room; delirium; failure of memory, and dementia. Insanity is occasionally met with in Graves' disease.

It appears that certain mental changes may accompany the different forms of valvular disease. The subjects of aortic disease are liable to attacks of excitement, with flushing of the face and possibly epistaxis, noisy anger, hasty rejoinder, and even destructiveness. Lunatics suffering from the same lesion are also peculiarly excitable, obstinate, and irritable; they struggle on the least provocation, have outbursts of anger, of abuse and violence, and may be dangerous. Hallucinations of vision are sometimes met with in aortic disease. General paralysis and tabes are also

not uncommonly accompanied by aortic valvular disease due to syphilis. Visual, auditory, and olfactory hallucinations are occasionally observed in patients with mitral disease; they have fits of depression and suspicion; and their memory for recent events markedly fails. In the insane, mitral lesions may be associated with melancholia, a grumbling morose disposition, and delusions of persecution and injury.

The occurrence of cerebral embolism or thrombosis should at once direct attention to the heart.

Dropsy.—Cardiac dropsy, that is, dropsy caused by disease of the heart, occurs in two forms: (1) œdema; (2) effusions into the serous sacs.

The pathology of this form of dropsy, together with the appearances which it presents and the symptoms to which it may give rise, will be fully considered subsequently.

Embolism.—Certain of the viscera, particularly the *spleen* and the *brain*, as well as the limbs, may suddenly present the clinical phenomena of arterial embolism of cardiac origin. Even the aorta is occasionally blocked by a large embolus.

GENERAL COURSE AND PROGNOSIS OF HEART DISEASE

A description of the physical signs and general symptomatology of diseases of the heart conveys but an imperfect conception of their clinical characters, unless it be accompanied with an account of the general course which they follow—that is, their commencement, progress, and different terminations. Along with this subject the prognosis or prospective course of cardiac disease will be conveniently considered.

In respect of their **course**, diseases of the heart fall into two groups, acute and chronic.

I. ACUTE DISEASES include acute inflammation of the endo-, peri-, and myo-cardium, caused by infections and toxins; acute parenchymatous and fatty degenerations due to the same pathogenetic influences, to anæmia, or to toxæmia; and strains of the walls or valves and wounds, the results of physical exertion or violence. The diseases belonging to this group have a definite, possibly sudden, commencement, and their characteristic clinical phenomena are rapidly developed. Their symptoms are active and often severe. At first they are those of increased cardiac action; upon these the physical signs of valvular and pericardial lesions, particularly murmurs, and of acute dilatation and possibly failure of the heart, soon supervene. Constitutional symptoms, including fever, are a common accompaniment. The progress of these acute cardiac diseases is comparatively rapid, and their duration short. They terminate either in death, which is unusual, but may possibly be sudden; in complete recovery not infrequently; but most commonly of all in permanent damage of the valves, walls, or pericardium, the lesions which originate in this acute way constituting one of the two groups of chronic heart disease which will be described presently.

The **prognosis** of acute disease of the heart—that is, the forecast whether its issue will be in death, in recovery, or in chronic cardiac lesion—must be based on as correct an estimate as possible of three sets of facts. The first of these are etiological: a knowledge of the nature of the cause that is at work, and of the resistant and recuperative powers of the individual patient. Thus the prospect of recovery is very small in infective endocarditis, in nephritic pericarditis, and in acute scarlatinal dilatation of the heart;

whilst a rheumatic origin in acute cardiac disease justifies the opinion that there is little risk of death, but, unfortunately, as little hope of complete recovery, the almost certain result being permanent valvular, myocardial, or pericardial lesion. The second group of data available for prognosis relates to the characters of the structural changes in the heart, including their seat, form, and severity, as well as to the state of the other organs, such as the lungs, pleura, and kidneys, which may be involved in the disease. For example, inflammation of the heart in acute rheumatism is far more unpromising when it involves the endocardium than the pericardium; aortic lesions are more serious than mitral; incompetence is more serious than obstruction at the aortic orifice; pneumonia, as an accompaniment of rheumatic endocarditis, is almost certainly fatal. The third set of elements of prognosis in acute cardiac disease includes the clinical characters, both local and constitutional—in other words, the symptoms and signs, particularly those which relate to the force, frequency and rhythm of the heart; and the clinical course. Evidences of embarrassment, and still more of failure, excessive frequency, infrequency, and arrhythmia always give rise to anxiety about the result. But the prognostic gravity of acute endocarditis lies not so much in its immediate danger to life, as in its liability to end in chronic valvular disease.

2. CHRONIC DISEASES of the heart constitute a far larger, more complex, and more important group than the acute. They originate in two ways. (1) Most commonly they are the *results of acute disease*, as has just been described. This is particularly true of chronic lesions of the valves—lesions left by acute morbid processes in early life, and now long ended, like a stiff joint or a shortened limb. (2) Other chronic diseases of the heart are *the results of slower, more continuous, or recurrent causes*. Such are valvular lesions produced by the physical stress of laborious occupations, and strains of the walls of the different chambers arising in the same way or from emphysema; valvular and parietal degenerations set up by the actions of such poisons as alcohol and syphilis, or that can be traced to disordered metabolism, as met with in gout, glycosuria, obesity, arterial degeneration, chronic nephritis, prolonged nervous strain, or the influences of advancing age. The commencement of cases belonging to this group is essentially insidious, in middle-aged or elderly persons. Their development is slow, but on the whole progressive, for their causes, unlike those of chronic diseases originating acutely, are persistent, nutrition is imperfect, repair is

of a low type, and the lesions are liable to spread and multiply. With these chronic diseases of the heart may be classed congenital affections.

The **clinical course** of chronic heart disease, in whichever of these two ways it may have arisen, comprises two phases. The one is characterised by the virtual absence of symptoms proper. Whilst the original structural damage remains, as proved by the physical signs, particularly endocardial murmurs, the attendant functional defect has been undone, made good, neutralised, or compensated by secondary changes in the walls of the heart; and the name of *compensation* is given to this phase of cardiac disease. The health and capacity for work of the subject of compensated heart disease may be equal to those of the average man; and it is not until some considerable change occurs, either, on the one hand, in the heart by fresh lesions or failure of nutrition, or, on the other hand, in the circumstances under which life is passed, that symptoms of inadequacy make their appearance in the circulation. The case then passes into the other or second phase, commonly known as *cardiac failure*. This is characterised by the phenomena of different degrees of the circulatory disability and disorder, the præcordial discomfort or distress, and the disturbances of the more closely associated organs, which have been described in a previous section. Under correct treatment these symptoms may diminish in severity and temporarily disappear—to return at a subsequent period under similar circumstances; and thus the course of chronic cardiac disease commonly consists of longer or shorter periods of comparative health, broken by repeated attacks of cardiac embarrassment or failure. If these occur in an aggravated form the case may terminate fatally, either by gradual cardiac failure with dropsy, or from some urgent complication, or it may be suddenly in syncope or angina pectoris. It will be understood from this description that compensation and failure are not sharply defined conditions; that compensation may be imperfectly established and insecure, and may readily pass then into failure; and that failure may similarly be of different degrees and of variable duration in different instances.

The remarkably favourable course which chronic disease of the valves and walls of the heart follows in so many instances, and for an indefinite length of time, is accounted for by the supervention of hypertrophy of the myocardium and dilatation of the cardiac chambers, automatic results of the disturbances of intra-cardiac pressure that had been produced by the lesions. Compensation is so important in respect of (i.) its origin, of (ii.) its maintenance,

and of (iii.) its temporary or ultimate failure, that it demands detailed consideration.

i. *Origin of compensation.* — Chronic valvular diseases of the heart, attended with obstruction or regurgitation at the different ostia, necessarily disturb the balance of the circulation. Whatever their form, they are calculated to underfill the arteries, to promote unnatural delay and accumulation of blood within the different chambers of the heart, and to permit venous over-distension. In other words, they favour abnormal distributions of the blood and of the blood pressure in the arteries, heart, and veins respectively. Chronic myocardial lesions and adherent pericardium obviously will have a similar effect. These lesions, however, prove to give rise to two attendant, or secondary, changes in the heart, which neutralise the effects of the primary damage on the circulation. The first of these is *hypertrophy of the muscular walls*, which meets the disturbed local pressure; the second is *dilatation of the chambers*, which provides accommodation for the local accumulations of blood. Similarly, increased peripheral resistance in arterial strain and degeneration, or in Bright's disease, automatically induces simple hypertrophy of the left ventricle; and increased pulmonic pressure in chronic pulmonary disease leads to enlargement of the right ventricle. By these natural provisions of physiological recuperation the disabilities of the primary lesions are corrected, and a condition of compensation is established. Its mode of origin is readily appreciated. *Compensatory hypertrophy* of the heart is the physiological result of increased intra-cardiac pressure or increased cardiac work. If an opening be stenosed, for example in aortic disease, or if the peripheral resistance be excessive, the rise of pressure behind the obstruction evokes increased action of the walls of the chamber, and the myocardium presently grows, thickens, or hypertrophies, in consequence of increased activity entailed by increased work, provided that the demand is not excessive, and that nutrition in the full sense of the term is sufficient. *Compensatory dilatation* of the heart is a physical result of over-filling of one or more of the chambers. It depends on extensibility of the walls, which yield and accommodate a larger volume of blood than ordinary: disturbance of blood distribution is met by the simple mechanical provision of making room for the excess by stretching of the walls. But the process cannot and does not remain of this simple kind. The capacity of the chamber being increased, the geometrical increase of internal pressure on its walls (see p. 299) and of the weight of blood to be discharged evokes increased force of systolic contraction, and in due course hyper-

trophy is developed—dilatation with hypertrophy. Hypertrophy is the necessary complement of dilatation from over-filling in a well-nourished heart.

Compensatory changes in the heart are not always limited to the walls of the chamber immediately in the rear of a valvular lesion or seat of increased resistance. Disturbances of pressure readily make themselves felt backwards—for example, from the left ventricle through the left auricle and pulmonic circuit to the right side of the heart—the degree to which they do so being in inverse relation to the completeness or sufficiency of the hypertrophy. Further, it must be observed that compensation of certain lesions is necessarily imperfect. Thus lesions at the mitral orifice can never be fully compensated by dilatation and hypertrophy of the left auricle, which has no valves behind it. The pulmonic circulation thus comes to be continuously over-filled—veins, capillaries, and arteries: the tension is abnormally high at the mouth of the pulmonary artery, as evidenced by accentuation of the second sound over it. It is the right ventricle that mainly effects compensation in mitral disease.

ii. *Maintenance of compensation.*—Thus originating and established, compensation is maintained for an indefinite length of time, possibly for many years, if certain conditions be fulfilled. These are: (1) a sufficient supply of healthy blood through the coronary arteries to the myocardium and all elastic structures of the walls, that is, abundant alimentation and oxygenation; (2) wholesome nervous influences through the trophic nerves; and (3) reasonable limitation of the demands for increased work made on the extensile and muscular tissues of the disabled heart, a condition which also implies that the lesion shall be of moderate severity. But it must be carefully observed that it is only under these favourable conditions that the subject of chronic disease of the heart remains healthy; for, although healthy, he is unsound, and, being unsound, the functional capacity of his heart is definitely reduced. A sound heart is constituted, and always prepared, to meet unusual stress and emergencies of other kinds by possessing a reserve of elasticity and of force-producing tissue. This reserve has been drawn upon, and possibly exhausted, in compensation; and, according to the demand on it, it may be near its limit. The compensated heart in valvular disease may readily meet the demands of walking on the level, of easy mental occupation, of moderate indulgence in food, of equable climatic conditions; but the subject of it may not be able to climb a hill, to undertake highly responsible work, to eat

an indigestible meal, or to catch cold, without suffering from disturbance of the circulation, although in a very great number of cases compensation is so complete that even these risks are run with safety, and health appears to be perfect. Thus compensation is a relative, not an absolute state, and, being conditional, it is uncertain and precarious as compared with soundness. Some of the subjects of valvular disease live constantly in a state of circulatory instability. Every considerable strain, of whatever kind, produces phenomena of cardiac embarrassment, such as dyspnœa, cough, palpitation, and præcordial distress, or it may be faintness or giddiness; and these are accompanied by disturbances of the impulse and rhythm of the heart and pulse. Compensation is threatened—indeed, for the time it is insufficient—and, exceptionally, death may occur suddenly; but more commonly, with the return of favourable conditions compensation is regained, and the “symptoms of heart disease” pass off. In peripheral obstructions such as chronic Bright’s disease and general arterial degeneration, as contrasted with valvular disease, the primary lesion is usually progressive, and the duration of compensation is definitely limited.

If we turn now to the subject of the general **prognosis**, we find that chronic disease of the heart is not a hopeless condition, as is commonly regarded. It is by no means necessarily fatal, or even incurable. Whilst the popular opinion of the prospect of life and health in this class of disease is that the patient is disabled for active occupations, that his life is uninsurable, and that death may occur suddenly at any moment, prognosis in disease of the heart, based upon a correct knowledge and use of the facts and considerations which have been reviewed in the previous sections, enables us to reach a very different conclusion. In by far the larger number of cases the lesion is compensated. Thus, life is not immediately threatened; and, so long as compensation continues, life will not necessarily be shortened. In cardiac disease with compensation prognosis relates to the maintenance of this state; in cardiac failure it relates to the recovery of compensation. Thus the first problem in connection with the prognosis of chronic heart disease is the prospect of maintaining compensation. In order to form a rational estimate of this there must be taken into account: (1) the *structural characters* of the cardiac disease, whether (a) *valvular* or (b) *myocardial*; (2) its nature or *origin*; and (3) the *circumstances of the individual patient*, particularly the conditions under which he is living and will live in future.

(1) In *structural disease* of (a) the *valves* the course which it will

follow, its duration, and the manner of its termination partly depend upon the *situation* of the lesion, whether aortic, mitral, tricuspid, or pulmonic; upon the *form* of the lesion, whether obstructive, incompetent, or both, at each of the orifices; and upon the *extent* of the damage to the valvular apparatus. Thus, aortic lesions as a class are less favourable than mitral; mitral stenosis is more unfavourable than mitral incompetence; aortic incompetence is relatively dangerous; and tricuspid systolic murmur is ominous prognostically because it is usually a manifestation of cardiac failure, but it may be expected to disappear if the failing heart respond to remedies. Some lesions are so slight that it can be confidently prognosed that they will never sensibly disorder the circulation. But here it is necessary to repeat the caution against attempting to estimate the severity of valvular disease by the characters of the murmur which it produces. A loud murmur is usually significant of a vigorous heart, of temporary anæmia, or of passing excitement, and not necessarily of extensive lesion; whilst a weak murmur is one of the evidences of failing heart, and may therefore be of more serious import. The condition of the cardiac walls is another essential element in prognosis: whether hypertrophied or dilated, or both. Pure hypertrophy is compensatory; it is accepted as an assurance of life and comparative health. At the same time, it may be an evidence of the existence of serious disease of some kind, whether cardiac or vascular: thus in its most perfect form, the left ventricular hypertrophy of chronic Bright's disease, it is prognostically of the worst omen, because it is a measure of the gravity of the renal and arterial lesions. Dilatation from overfilling, with associated hypertrophy, whilst conservative, is also a more precarious condition.

(b) *Chronic degenerative changes in the myocardium*, fatty and fibroid, being commonly associated with intractable disease of the coronary arteries, offer a much less favourable prognosis. In this connection a careful estimate must be made of the state of the other viscera, particularly of the arterial system generally, of the kidneys, and of the lungs.

(2) Diseases of the heart present a different prognosis according to the *origin, nature, or kind* of each.

Rheumatic lesions are relatively favourable because they are nothing more than the results of repair, which has distorted or stiffened a segment or obstructed an orifice. They are essentially non-progressive, and it is important to observe that the longer they have been known to exist with good compensation, the better is the

prognosis. Still, when they involve the auriculo-ventricular rings, the possibility of gradual and serious increase of the resulting stenosis must not be overlooked. Moreover, valves so damaged are predisposed to the incidence of malignant endocarditis. *Syphilitic* valvular disease is a dangerous process, possibly associated with active disease of the coronary arteries and myocardium: life is relatively short and quite insecure; death may be sudden. In valvular *atheroma* a correct forecast is difficult, but on the whole unfavourable. This kind of lesion is the result of imperfect nutrition, due either to disorder of the blood, or to disease of the coronary arteries, or to both; and, whilst not peculiarly dangerous in itself, is probably accompanied with *degeneration* of the myocardium, which is similarly impoverished, and with unsoundness of the arteries of the other viscera, particularly the kidneys. The result to be anticipated, therefore, in a considerable proportion of these cases is either sudden death, or slow failure of the heart peculiarly resistant to treatment; or, as often happens, death from cerebral hæmorrhage or chronic nephritis. In mechanical *injuries* of the cusps, chordæ, or other parts of the valvular apparatus, and in dilatation of the cavities as the result of acute or chronic strain, the resulting circulatory disability is to be regarded as permanent, although of very different degrees in different instances, according to the severity of the lesion.

(3) A knowledge of the *circumstances of the individual patient* is all-important as a guide to prognosis, because they constitute the influences under which the heart will have to work, including the dangers that threaten it, and will in great measure determine how long compensation can be maintained. They include the patient's age; occupation; his social position and means; his disposition and habits; his food and other ingesta, including stimulants; the nervous influences of every kind around him; the different diseases, acute and chronic, to which he is exposed, particularly such as involve the circulation, like chronic granular kidney; the strains of growth and development in the young, and of special functional activity in women; and the advent and progress of old age. Whether the lesion be aortic or mitral, obstructive or incompetent, whether rheumatic, syphilitic, or otherwise, the future of it in great measure obviously depends upon the conditions in which the patient is expected to live.

iii. *Failure of compensation*.—Occasionally the conditions necessary to compensation are insufficient from the first, and the process fails to be established; far more often they become insufficient, and then it fails to be maintained. Speaking generally, to begin with,

failure occurs from either of two sides. The compensated heart is either insufficiently nourished or it is excessively taxed. On the one hand, nutrition is insufficient. The coronary blood-supply may have become defective in quality or in quantity, as a consequence either of general anæmia from want or from waste, or of local vascular disease; or the blood may be poisoned by toxic substances of extrinsic or intrinsic source; or the trophic nerves of the myocardium may be disordered, whether directly or reflexly. On the other hand, the demands on the walls may have grown excessive, by increase of valvular lesions; or more commonly by the requirements of growth and development, as in adolescents, or of fresh functional activity, as in married women; or by increase of peripheral resistance—from muscular strain, bronchitis, gout, senile changes in the arteries, or Bright's disease. Very commonly several of these disturbances occur together, as in the poor, who are ill-fed, distressed, over-worked, and often unsound. These and other individual causes of cardiac failure demand closer consideration; and they are most naturally and conveniently discussed in connection with each of the principal periods of life, when the prognosis has to be estimated.

In *childhood* compensation is commonly threatened by acute diseases, anæmia, impoverishment as the result of improper feeding (whether insufficient or excessive), hardship, and bad management generally. At the same time, whilst this age, as being the period of active growth of the heart, is favourable to compensatory hypertrophy, the increase of size and development of the body is a severe physiological strain on the circulation; and the prognosis of cardiac disease is relatively more serious in the child. In *boyhood* and during *adolescence*, fresh rheumatism or chorea is an ever-threatening danger; and physical over-exertion and mental strain are influences that have always to be reckoned with, in connection with the cardinal questions of exercise and education.

During the next period, extending *from twenty-one to forty-five*, the prognosis of compensated disease of the heart is partly based on the anticipation of another series of influences which threaten compensation. The danger of rheumatism and chorea has in great measure passed; exercise is still likely to be abused; but more important causes of cardiac disturbance have now to be feared. These are: in men, their occupations, alcohol, tobacco, and syphilis; in women, the risks connected with maternity in its different phases; whilst in both sexes it is between the ages of twenty-one and forty-five that the course and promise of the

patient's life, whether fortunate or unfortunate, comfortable or hard, as well as his disposition and character, are practically settled, and therewith the prospect of maintaining compensation is to be estimated accordingly.

From forty-five to seventy-five, the most unfavourable influence on the diseased heart is progressive degeneration. Involving the arterial system generally, it strains the cardiac walls; when it affects the coronaries in particular, the valves become atheromatous and the myocardium is impoverished, weakened, and diseased. But to ensure a correct prognosis at this age vascular degeneration must be traced to some cause, which may or may not be remediable: to gout, glycosuria, alcoholism, Bright's disease, syphilis, physical stress, or nervous exhaustion.

Failure of the heart is, as a rule, developed gradually. The objective and subjective symptoms of distress and dropsy (p. 370) increase slowly, steadily or intermittently, according to circumstances. Sometimes they are arrested by prompt and correct treatment; and they may be indefinitely removed if recovery have been thorough and the causes of its occurrence be faithfully controlled. In other instances cardiac failure advances to death, by excessive dropsy, hæmorrhage, and exhaustion; and in most cases general visceral deterioration eventually sets a limit to existence considerably short of the natural term, in spite of temporary and perhaps repeated improvement. Less commonly cardiac failure is developed rapidly—*acute dilatation with failure of the heart*; and the student will now be able to understand how this grave condition may occur as a primary lesion, that is, independently of previous compensatory enlargement, when the myocardium, enfeebled by acute degeneration, as in scarlet fever or typhoid, fails sufficiently to meet the increased peripheral resistance produced by renal complications or premature exercise.

Failure of the heart essentially consists in failure of the ventricles to complete their output. The force of systole—of the left ventricle, for example—is exhausted before the chamber is sufficiently emptied, and a residue is left undischarged. In the next diastolic period the charge received is added to the residue, and the chamber is over-distended or dilated—dilatation in consequence of accumulation from insufficient emptying due to failure of the myocardium. *Dilatation from failure* is an entirely different process in nature and in gravity from *compensatory dilatation*, which is a simple physical effect of overfilling. Further, once commenced, dilatation from failure increases and works back-

wards, either by stretching the walls of the ventricle until the auriculo-ventricular valve becomes incompetent and the auricle is flooded, or by raising the pressure so high in the ventricle as to interfere with the systole of the auricle, which, in its turn, is insufficiently emptied. Thus, failure of the left ventricle travels backwards through the lungs (rise of pulmonic pressure, congestion, œdema, hæmorrhage, dyspnœa, cough and hæmoptysis, as described) to the right chambers, which are successively dilated, and thence to the great veins (distension, pulsation), to the viscera (mechanical congestion and functional disorder), the systemic radicles (lividity, dropsy), the serous sacs (effusion), and the mucous surfaces (catarrhs, fluxes, and hæmorrhages). Thereupon either removal of the cause of failure by means of rest, feeding, etc., or, on the other hand, artificial increase of the cardiac force by means of food, stimulants, and drugs, or both sets of measures together, or these along with the establishment of artificial relief of the venous pressure by bleeding, purging, and paracentesis, will often arrest the process of failure, reduce the accumulations, and *restore compensation* for an indefinite period. But failure is likely to return sooner or later, and it finally proves fatal.

The **prognosis** of cardiac failure, that is, the prospect of restoring compensation, is estimated by the same facts as are employed in the prognosis of cardiac disease with compensation, and by the clinical phenomena. In addition to the *structural characters*, and the *origin* of the primary lesion, which have to be weighed with the same care as before, the *circumstances that have now undone compensation* must be particularly regarded, inasmuch as some of these are temporary or removable, with fair prospect of recovery of compensation, whilst others are permanent or progressive, and present no hope in this respect. Thus rheumatism and chorea and many of the acute diseases, including bronchitis, respond to treatment, and the cardiac failure which they have induced passes off. Physical overwork and impoverishment, perhaps the commonest of the causes of cardiac failure in the poor, can be temporarily removed by rest, warmth, and nutritious diet, and with them the dropsy and other manifestations of failure of the heart often temporarily disappear. Such nervous causes of loss of compensation as worry, misfortune, and mental strain are much more difficult to control, and the prognosis of cardiac failure due to them is correspondingly unfavourable. Still more unfavourable, because entirely irremediable, is Bright's disease as an insidious cause of failure of the heart in chronic rheumatic lesions when the patient

reaches middle life ; while gout and other serious disturbances of metabolism, such as glycosuria and obesity, are scarcely more favourable. It will thus be seen that in every instance the circumstance of the patient's life that is the immediate cause of myocardial failure and consequent mechanical congestion of the viscera, dropsy, and albuminuria, must be faithfully ascertained and duly weighed before a correct prognosis can be reached.

Many of the *clinical phenomena* are useful prognostic guides. Although individual symptoms and signs, such as pain (including angina pectoris), faintness, palpitation, præcordial distress, weak apex-beat and murmurs, are more or less common to all cases of failure and to all kinds of cardiac disease, and relatively of uncertain value as elements of prognosis, it is correct to say that the more severe the patient's sufferings as a whole, and the greater the disturbance of the circulation and associated functions, the more unfavourable is the prospect. Urgent præcordial pain and oppression, paroxysms of dyspnoea, anxiety, pulselessness, faintness, and cold sweats, cyanosis, somnolence and weak delirium—especially in combination—are grave symptoms. Of physical signs, the worst are increasing weakness of the impulse, extension of the præcordial dulness transversely in both directions, asystolism, the disappearance of a structural murmur, such as mitral presystolic, and the development of a tricuspid systolic murmur, visible reflux in the cervical veins, and pulsation of the liver ; and along with these great frequency, irregularity, and feebleness of the radial pulse. Particularly ominous is the occurrence of pulmonary or of splenic infarction. Of all the strictly clinical means of prognosis at our disposal in cardiac failure, the most exact and at the same time easy is the amount of urine, by which the vigour of the heart, the effect of treatment, and the progress of the case can be accurately estimated. A clinical history of similar attacks of cardiac failure with dropsy, which were relieved only by tapping, gives an unpromising aspect to a case ; yet patients have been successfully treated for cardiac dropsy many times in succession over a number of years, particularly where the cause of failure was poverty and overwork. As will be presently described under "treatment," little improvement can be expected for the first two or three days of cardiac failure. Promising events then are the moderation of the different symptoms just mentioned, increase in the amount of urine, which is often great and almost sudden, decline of dropsy, and return of the physical signs and pulse characteristic of the primary lesion. Unfortunately, in some instances the medicinal remedies employed

not only fail to produce these favourable results, but actually disagree with the patient and aggravate his condition by causing vomiting, so that they have to be intermitted or abandoned, and food also can be taken only with difficulty. The prognosis then becomes grave. But the clinical rule is not to despair under these circumstances, nor of any case, however severe. Recovery may take place after all the symptoms and signs described as unfavourable have been fully developed.

In those kinds of chronic disease of the heart which essentially consist or originate in failure of nutrition from coronary disease, compensation is impossible. The structural conditions of the myocardium in atrophy and fatty degeneration are essentially negations of compensatory hypertrophy; and the course which these diseases follow is one of chronic cardiac asthenia interrupted by attacks, on slight provocation, of acute cardiac distress or failure, which may be very alarming or even immediately fatal.

THE PRINCIPLES OF TREATMENT IN DISEASE OF THE HEART

This subject will be most profitably discussed in the order of the natural history and course of disease of the heart in general, as presented in the last section: viz. (1) acute cardiac disease; (2) chronic cardiac disease with compensation; (3) failing compensation and cardiac failure. Thereafter there will have to be noticed: (4) the general treatment of those kinds of chronic disease of the heart which originate in insidious degeneration in older subjects.

1. The objects to be kept in view in the treatment of *acute* inflammation of the heart and pericardium are mainly two. The first or immediate object is to prevent or remove acute failure of the myocardium, and to relieve disorder and distress. The second or remote object is to promote early, rapid, and complete resolution of the morbid process, in order to prevent chronic valvular disease, parietal fibrosis, and pericardial adhesion; or, should the attempt fail, to moderate the damage of the valves and the disabilities of the cardiac walls. With these ends in view, the inflammatory process must be controlled from two different sides. On the one hand, its destructive agent or factor, that is, the extrinsic cause of the disease, has to be dealt with directly—rheumatism, for example, by means of the salicyl compounds, septicæmia with a specific serum. These remedies are given at once and in full doses; they are not discontinued until the symptoms have been subdued for several days; and they are resumed if relapse should occur, as happens not infrequently. On the other hand, an effort is made to influence the intrinsic elements of the inflammation, including the local conditions of repair. These are of exceptional importance in connection with the heart. The heart never enjoys complete rest, an essential condition of successful healing. Its valves and associated apparatus are subjected to the stress of movement and tension. It is constantly liable to be disturbed by changes in the blood-pressure; by the state of the neighbouring organs, particularly by distension of the stomach and intestines; and by the many influences which reach it through the nervous system. These serious drawbacks to resolution and repair in acute cardiac disease demand the absolute avoidance of exertion and excitement during the whole course of the illness and of con-

valescence ; a digestible diet of moderate amount ; strict regulation of the bowels ; and faithful nursing, assisted as may be necessary with medicinal remedies. Whilst the etiological and pathological indications are thus attended to, urgent symptoms and signs of myocardial embarrassment or actual failure necessitate the employment of other measures, to sustain the heart, to unload its over-distended chambers, and to relieve distress, such as diffusible stimulants, strychnine and digitalis, the abstraction of blood, and possibly morphine.

2. When the acute stage has been safely passed, and health and strength are being restored, a new set of therapeutical indications demands fulfilment to meet the consequent *chronic* condition. We shall assume that permanent mitral incompetence remains as the result of acute endocarditis. Compensation is being naturally established, by dilatation with hypertrophy of the left auricle and left ventricle, and by hypertrophy of the right ventricle ; the pulmonary circuit is under permanent high pressure. The therapeutical indication pointed by a consideration of this new adjustment of work and effective forces is to promote it ; and, having helped to establish compensation, to maintain it. We know the conditions of compensation, and these we endeavour to secure. The diet must be highly nutritious and digestible, whilst strict attention is paid to elimination. An effort is made to secure fresh air and sunshine during convalescence ; and hæmatinic medicines are prescribed. The unfavourable influences of educational or business worry must be prevented. The demands that can be safely made on the muscular and elastic walls of the heart have to be estimated in each case, and definite instructions given as to work, exercise, and play. In a word, compensation is established by making the circumstances of the patient's life as hygienic as possible in every respect, and by preventing an increase of the lesion or other cause of strain of the myocardium. Hypertrophy itself, whether as a process or as a state of health, need call for no drugs. Attacks of dyspnœa, palpitation, faintness, or præcordial distress occurring in the course of compensation should be traced to their cause in each instance—excitement, exertion, dietetic errors, a hot unwholesome atmosphere, or the abuse of alcohol, tobacco, and the like ; and this dealt with. Direct treatment of this order may be assisted with ether, alcohol, ammonia, and other anti-spasmodics and carminatives, as well as with bromides and nitrites ; but the primary indication must never be forgotten. Either warm or cold local applications to the præcordia often prove useful.

3. We next approach the phase of threatening or actual breakdown of compensation by dilatation with *failure of the heart*, probably after years of fair or even good health. One or other of the unfavourable influences of daily life falls upon the subject of chronic valvular disease. We have seen that the principal of these are impoverishment or poisoning of the nutrient (coronary) blood, physical stress, nervous excitement or depression, pulmonary affections, increase of the cardiac lesion, periodic perturbations of the circulation, and the advance of age. The first indication for treatment of failing heart is to discover which of these or other possible causes is at work, and to deal with it direct. Overwork, worry, and mal-nutrition, separately or combined, being most commonly to blame, especially in the poor, the routine practice is to put the patient to bed and diet him correctly, and this is usually successful; but it must not be forgotten that the opposite line may have to be followed—for instance, in those patients who have undone compensation by sedentary living and over-indulgence in food, alcohol, and tobacco.

Whilst thus attending to the cause of failure, we have to fulfil certain great pathological indications, impressed upon us by a study of residual dilatation of the cardiac chambers. These are: (*a*) to increase the force of the muscular walls, in order to obtain effective systolic out-put; (*b*) to lower high arterial tension, should such exist; and (*c*) to restore the normal venous pressure.

(*a*) The first of these objects, the development of increased cardiac force, is effected by means of food, of stimulants, of drugs which act favourably on the circulation, and of various methodical muscular exercises that improve the tone of the cardiac muscles. A selection is made from these different measures according to the urgency of the case. Food is necessarily always ordered, varying in kind, form, and amount, according to circumstances, as will be discussed presently. If the symptoms be severe, rapid cardiac stimulants are demanded—ether, alcohol, ammonia, strychnine subcutaneously, or nitrites. The digitalis group of drugs acts more slowly, as powerfully, and far longer, upon the heart, by increasing the vigour of systole, and thus sufficiently emptying the ventricles, at the same time lengthening diastole and thus emptying the veins and the auricles—in a word, by producing a full and complete cardiac cycle. The tests of their success are not only relief of præcordial distress and improvement in the pulse, but marked diuresis and disappearance of dropsy. Methodical exercises are specially suitable to conditions of

threatening or incipient failure caused by sedentary habits and high living.

(*b*) The second indication for the relief of residual dilatation is to reduce, if necessary, peripheral resistance, in both pulmonic and systemic circuits, in order to facilitate systolic out-put from all the chambers—that is, the passage of the blood through the heart. Rest is the means commonly employed. Bodily rest is made as complete as possible, the patient keeping his bed with the head and shoulders raised, or sitting in an easy-chair with the back and arms well supported, in order to favour respiration. Mental quiet is essential. Next to rest, purgation has the effect of lowering arterial tension both directly and by relieving venous congestion; and iodides and nitrites, vascular depressants of different degrees and rapidity, produce this effect, whilst digitalis and its congeners, in the third stage of their actions, relax the renal arterioles.

(*c*) The remaining indication for unloading and thus assisting an over-distended heart, though the last in order of systematic description, should be the first to receive attention. It is to remove the accumulated residues in the veins and the mechanically congested viscera, and the dropsical collections behind them. If the condition be urgent, abstraction of blood is practised by means of venesection, cupping, or leeching. Less swift, but far more often appropriate, is free purgation with hydragogue drugs, such as compound jalap powder, elaterin, colocynth, and salines following on calomel or other mercurials. The improvement obtained from such cathartic measures, repeated daily if necessary, is very great; and at the same time they relieve in particular the portal system, and thus promote the recovery of the alimentary organs—a vital point, as nutrition is restored—and remove the grave impediment to cardiac action of flatulent distension.

Instead of draining off by the bowels and kidneys the serous accumulations which constitute cardiac dropsy, we may do so directly by means of paracentesis of the serous sacs or subcutaneous tissues. This is a thorough method and a highly successful one, provided that antiseptic precautions be faithfully observed. Paracentesis is often a matter of urgency, and in the same way it is of twofold benefit when the pleural or the peritoneal cavity is emptied and the heart relieved of a physical as well as of a physiological burden.

If the indications already discussed be strictly fulfilled in a tractable case of cardiac failure, the many kinds of distress that accompany it will automatically disappear. But as time is a

necessary factor of the method of treatment, various *palliative* measures are usually demanded for a few days. The cardiac and respiratory stimulants already mentioned—strychnine subcutaneously, ether, ammonia, and alcohol—are given for dyspnœa, whether continuous or paroxysmal. Occasionally it may demand immediate paracentesis of the chest or abdomen, or even venesection. Obviously also dyspnœa is a call for freedom to breathe, which is best afforded in an “easy” chair with the feet on the floor and the back and arms well supported. Evacuation of the bowels and the avoidance of flatulent food are equally necessary. The same series of remedies affords relief from præcordial distress, pain, palpitation, and faintness; and they are always assisted by cheerfulness and reassurance on the part of the doctor and nurse. Paroxysmal cough, dyspnœa, and hæmoptysis, accompanied by the signs of pulmonary congestion, are rationally treated with free leeching. Insomnia may require for a time to be met with sulphonal, paraldehyde, urethane, or chloralamide, or with a combination of morphine and strychnine hypodermically.

The relief of digestive disorders in cardiac failure, and of the serious interference with the action of the heart to which they give rise, is best studied in connection with the all-important subject of *diet*. The objects to be kept before us in dieting a case of cardiac failure are mainly three: to nourish and invigorate the myocardium; to prevent, and if necessary remove, indigestion, particularly flatulence; and to limit the amount of fluids added to the blood and lymph. Food accordingly must be highly nutritious and attractive, easily digestible, and as far as possible solid. Should there be nausea, vomiting, and other evidences of inability to take, retain, or digest solid food, then essences, jellies, and strong infusions of meat, and peptonised milk and cereals, with or without a few drachms of brandy in alkaline water, or egg-flips, are to be given, whilst the mercurial, saline, or other purgatives are relieving the mechanical congestion of the portal system and gastro-duodenal catarrh; but the patient must not be ordered large “feeds” of beef-tea and milk at short intervals, as in typhoid fever. Ordinarily, however, light solids can be taken easily—eggs, or bacon, or fresh white fish and stale bread for breakfast; lean and tender mutton with bread, spinach, and a farinaceous or custard pudding at mid-day; a small cup of weak fresh tea with bread-and-butter in the afternoon; and grilled or boiled fish, sweetbread, tripe, or a peptonised dish for supper. During the night a little fluid nourishment is permissible in the event of wakefulness; and either a cup of tea or two

drachms of brandy in water is a useful stimulant about 6 or 7 A.M. Drinks and other forms of refreshment between meals should be discouraged. Nearly still soda-water with a little stimulant is the best beverage at meat meals. Any further disturbance of digestion should be unhesitatingly met with mercurial purgation and reduction in the amount of food, as little changed as possible in its form ; not by having recourse to slops.

As the condition of the circulation improves, treatment is modified. Rest is maintained, both of body and mind, but all the medicines are given in reduced amount, and the range of diet is extended, the pulse, physical signs, symptoms, and state of the urine being closely watched as guides. The indication now is to re-establish compensation, and to do so with thoroughness so as to maintain it. Time is essential. For weeks or even months comparative rest is continued, varied more and more with regulated exercise, judicious dieting, and cardiac tonics, such as small doses of digitalis, strychnine, iron, and it may be arsenic.

4. Remedial treatment of *chronic myocardial disease*, in which the wall of the heart has undergone either atrophy as a whole, or fatty degeneration in its individual fibres, is usually impossible. The necessary conditions of hypertrophy, and indeed those of ordinary nutrition, are essentially deficient, whether from anæmia, toxæmia, or local vascular disease, or possibly from disturbance of the trophic influence of the nervous system. All that can be done is to search for the cause of nutritional failure, and to attempt to deal with it ; to reduce the functional demands on the walls of the heart to the level of the lessened capacity ; and to avert or relieve failure. If the cause of degeneration prove to lie in defective quality and quantity of the blood in the coronary circulation, it can sometimes be met with a carefully selected diet, fresh air, and hæmatinics like arsenic and iron. Chronic toxæmia may yield to the same classes of remedies, and to the ordinary treatment for gout, obesity, glycosuria, and other forms of chronic metabolic disorder which may have given rise to it. Exercise, whether of an ordinary kind in the open air, or in the form of methodical movements, with or without baths, is calculated to invigorate the myocardium, provided care be taken to employ it in suitable cases, and to avoid sudden or severe efforts. At the same time the nervous influences acting on the circulation, particularly those connected with the patient's occupation, must be closely supervised. Palliative treatment is directed to the prevention or the removal of cardiac embarrassment and distress, in the form of syncope or

angina. The first of these ends is most successfully attained by controlling the determining causes of such attacks, particularly exertion, excitement, and indigestion. If an attack should occur, it may be relieved by means of the nitrites or other drugs that rapidly stimulate the heart and lower the peripheral resistance.

DISEASES OF THE ENDOCARDIUM

ACUTE ENDOCARDITIS

Etiology.—Acute inflammation of the lining membrane of the heart is one of the effects of the action of infections. Of these the rheumatic poison is the most common. Natural resistance to these pathogenetic influences fails in young subjects, in hearts already damaged, and in the parts of the endocardium subjected to greater stress—the valves of the left ventricle—which are peculiarly liable to become inflamed under the determining influences of exertion and excitement.

Anatomical characters.—These have been described at p. 327, where it has been shown that whilst the inflammatory process rarely proceeds to destruction, repair often leads to permanent valvular defects.

Symptoms.—The constitutional symptoms observed in acute endocarditis are those of the acute febrile processes with which it is associated. In a large proportion of cases the disease is a manifestation of acute rheumatism (see Vol. I. p. 291). The local symptoms, which are insignificant in proportion to the gravity of the malady, are at first due to cardiac excitement; later on to cardiac failure in severe cases. The most important and only characteristic clinical phenomena are the physical signs. These include temporary increase of præcordial impulse, increase of præcordial dulness, and various changes in the cardiac sounds, which become rough, muffled, or blowing, or are accompanied by or converted into murmurs of which the mitral systolic is the most frequent.

The clinical course of acute endocarditis is variable; its duration is indefinite; and it terminates either in complete recovery, in incomplete recovery, *i.e.* valvular damage, or in death, which is very rarely due to it directly. It may be complicated with acute pericarditis and myocarditis, or other local manifestations of rheumatism, of which it has been itself usually described as a "complication." Occasionally it leads directly to arterial embolism.

Diagnosis.—The diagnosis of acute endocarditis turns on the development of a bruit under observation in the course of an acute febrile disease or chorea. This murmur may be confounded with

that due to chronic valvular lesion, unless the size of the heart, the condition of the circulation as a whole, the history of the case, and the physical signs from day to day be carefully regarded. It is also simulated by associated hæmic murmurs (p. 348).

Prognosis.—The prognosis of acute cardiac disease in general has been already discussed (see p. 376), from which it may be seen that in acute endocarditis the immediate forecast is favourable, but the remote prospect bad, since it terminates so often in valvular damage. In a few instances endocarditic murmurs disappear after some weeks or months.

Treatment.—Acute endocarditis is to be *averted* mainly by preventing acute rheumatism, particularly after one attack; and, if this do occur, by treating it with despatch and thoroughness.

The first indication for the *remedial* treatment of acute endocarditis is to deal with the cause, rarely other than the rheumatic poison, by means of specific drugs, of which the salicyl compounds are the chief—sodium salicylate or salicin in 15 to 20 grain doses, given every four hours until the temperature falls to normal, and then very gradually reduced during a period of several days. From the pathological characters we learn to reduce the force and frequency of the heart, in order to save the valves from violent agitation and tension, and promote repair. These objects are to be secured by means of bodily and mental (emotional) rest, assisted if necessary with anodynes, hypnotics, and other cerebral sedatives, of which morphine, employed with scrupulous care, is the most valuable. With the same end in view, the occurrence of flatulence and consequent disturbance of the heart are prevented by ordering the most digestible kinds of fluid food, such as milk and beef-tea, in moderate amounts. We have little direct control otherwise over the morbid process of endocarditis. Still, early treatment, close supervision throughout, and finally prolonged rest during convalescence, are often sufficient for the attainment of the primary object in view—to prevent a permanent lesion, or, at least, to minimise it. At the same time, different forms of præcordial distress may demand *palliative* treatment. This consists as much in preventing or removing injurious influences, such as excitement, movement, or improper feeding, as in the use of direct cardiac sedatives and antispasmodics, such as ether, ammonia, bromides, and morphine.

INFECTIVE ENDOCARDITIS

This disease is described in Vol. I., p. 77.

CHRONIC VALVULAR DISEASE

Etiology.—Chronic valvular disease of the heart is the result of acute endocarditis, degenerative processes, acute or chronic strain, excessive arterial pressure from other causes, and syphilis. Predisposition to cardiac disease from such influences respectively is necessarily greater at certain ages, in connection with different occupations, and under a variety of other circumstances.

Anatomical characters.—These will be found described in the section on general pathology under “Inflammation,” p. 326, and will be referred to here only in respect of their clinical bearings. The *seats* of disease are any or all of the valves, but far more frequently the mitral and aortic. The *forms* of damage, speaking broadly, are: (1) stenosis of the openings, which produces obstruction; (2) dilatation of the ostia, or other changes preventing exact apposition of the segments, which permits incompetence with regurgitation; and (3) both. Thus the following varieties of valvular lesions are met with:—mitral incompetence (30.5 per cent); mitral obstruction (16 per cent); double mitral disease (12 per cent); aortic obstruction (2 per cent); aortic incompetence (7.5 per cent); double aortic disease (12 per cent); different combinations of mitral and aortic lesions (19 per cent); tricuspid incompetence (from actual valvular lesion, as distinguished from simple dilatation of the ostium), tricuspid obstruction, pulmonic obstruction, and pulmonic incompetence, together only about 1 per cent; and very rarely combinations of these right-side lesions with the others. The *kinds* of lesions are: fibrotic, cicatricial, and other effects of the repair of acute endocarditis; chronic endocarditis; atheroma and syphilis associated with changes in the nutrient arteries; and mechanical injuries, etc.

The walls of one or more of the chambers are enlarged—hypertrophied, dilated, or both, in various associations; and the myocardial tissues may be found sound, congested, granular, fatty, or fibroid, according to circumstances. The condition of the other viscera varies according to the mode of death; in a large number of instances they are mechanically congested, and the tissues and serous sacs dropsical.

General symptoms and course.—Chronic valvular disease of the heart presents two widely different classes of clinical characters, according to its phase.

In the first phase, the patient is healthy although he is unsound. He confesses to certain disabilities of respiration, circulation, and

muscular vigour, different in different instances, and according to variable circumstances, when the heart is taxed by exertion or excitement; but he is not ill, and probably comes under observation with some other complaint. This is *valvular disease with compensation* (p. 378). Physical examination reveals the signs of cardiac enlargement and of quiet cardiac action, and one or more endocardial murmurs, with various associated changes in the cardiac sounds. The other viscera, the subcutaneous tissues, and the serous sacs present trifling, if any, evidence of circulatory failure. This condition may continue indefinitely for years, a life of perfect or at least fair health and activity being enjoyed, provided the person's circumstances are favourable.

The second class of clinical characters points to the occurrence of *loss of compensation*. The patient is seriously ill, suffers from dropsy, and complains of dyspnœa, præcordial distress or actual pain, palpitation, faintness, and many disturbances of the digestive, assimilative, urinary, and cerebral functions. Clinical examination elicits the symptoms and physical signs of *failure of the heart* (p. 370). Cardiac failure in chronic valvular disease may terminate in death; but in many instances it passes off, most probably to return sooner or later, according to the patient's circumstances, and with similar results.

Between the two extremes of compensation and failure there is a long series of different clinical conditions in chronic valvular disease. There are many degrees of completeness of compensation or of incompleteness of compensation—that is, of cardiac inadequacy—according to the kind, form, and seat of the valvular lesion, the condition of the walls of the heart, the state of the other viscera, and the circumstances of the patient's life. Common events are dyspnœa and cough on exertion, with possibly even a little hæmoptysis; faintness and præcordial oppression in mental excitement; attacks of orthopnœa, anxiety, and faintness during the night after a hearty meal; and transient œdema of the ankles. A degree of anæmia and vensity of the blood is seen in the integuments of the face and extremities and in the visible mucous membranes; the physical signs and pulse are characteristic of slight cardiac failure (p. 371). Such conditions of incomplete, precarious, or *unstable compensation* may vary rapidly and frequently from time to time in either direction; occasionally the patient dies suddenly; more frequently they end either in improved compensation or in failure with dropsy.

Rupture of the valves requires special reference. It is the result

either of severe muscular effort or of trauma ; and is attended with sudden præcordial distress, palpitation, dyspnœa, faintness, and possibly the development of an auto-audible murmur. Death may occur immediately, or the ordinary clinical characters and course of valvular disease may be developed.

VARIETIES OF VALVULAR LESIONS IN FORM AND SITE

MITRAL INCOMPETENCE.—This is the most common variety of chronic valvular disease. It is usually the result of acute endocarditis—generally rheumatic ; less often of degeneration or acute or chronic stress. It is also a consequence of dilatation with failure of the left ventricle in aortic and arterial disease, or in acute or chronic debility from atony of the muscular ring of the mitral orifice. The anatomical characters of valvular lesions giving rise to incompetence are described at p. 330. In connection with the mitral they present two principal forms. The first of these consists of vegetative growths, adhesions, shrinking or other irregularity of the cusps and chordæ, the result of acute endocarditis or of chronic or acute strain. The second form consists in dilatation of the ostium—whether this be, on the one hand, absolute, as the result of atony or of stretching of the ring ; or, on the other hand, relative only, an effect of dilatation of the left ventricle in cardiac failure and consequent disturbance of the valve-segments and their apparatus, which prevents their accurate apposition in systole. The heart is enlarged. The left auricle is dilated and hypertrophied, being filled from two directions ; the left ventricle is likewise dilated and hypertrophied, being overfilled and overweighted by the extra output of the left auricle. The right ventricle is hypertrophied at first, in response to increased pulmonic tension, but soon undergoes dilatation from insufficient emptying, which increases with failure ; and under the same circumstances the tricuspid orifice and the right auricle also yield. The pulmonic circulation is continuously under increased tension ; its capillaries are dilated and occasionally ruptured, and the branches of the pulmonary artery may be atheromatous. If the disease prove fatal, the systemic veins and the viscera present the characters of mechanical congestion in their full development, and the tissues and serous cavities are dropsical.

The **symptoms** of mitral incompetence, whether with perfect compensation, with imperfect compensation, or with failure, respectively, are in the first place typical of valvular disease in general.

More particularly, the effect of this lesion of the mitral valve is to embarrass the pulmonic circulation and the breathing; to interfere with the venous flow; to produce a dusky congestion of the features; and to impoverish the arterial circulation. Yet life may be maintained for years with fair health and activity, according to the nature and severity of the lesion and the patient's circumstances. Compensation is specially undermined by influences that fall on the lungs and right side of the heart (which mainly effects compensation), such as climate, season, and physical exertion; and it may often be restored by means of rest, warmth, and attention to digestion and elimination assisted by drugs. Death usually occurs with dropsy, but it may be sudden. If the lesion be degenerative, recovery is less common. Mitral incompetence from myocardial relaxation, or from dilatation secondary to aortic disease, passes off if the ventricle regain its vigour.

In mitral incompetence with compensation the **physical signs** are distinctive. The præcordia are often prominent, the impulse extensive and complex. The apex-beat is felt, and may be seen, to be displaced outwards and downwards, its precise seat varying with the degree of enlargement; its characters are not special, and it may be accompanied by systolic thrill in the same area. Occasionally there is systolic relapse. Præcordial dulness corresponds, and is also increased across the lower end of the sternum and over the right cartilages beyond it. The characteristic auscultatory signs are: (1) the mitral systolic murmur (p. 349), which is of different qualities according to a variety of circumstances, particularly the nature of the lesion, and is sometimes audible only in recumbency, with or without the first sound; and (2) accentuated second sound in the pulmonic area. The rhythm is often irregular. Very rarely the murmur disappears permanently. When the heart fails, the physical signs change (p. 371), the murmur either being lost or occurring only at short intervals with the stronger systoles in the arrhythmia. The pulse is usually small and of variable tension, the pulse-wave is irregular, frequent, short and weak; but it may not be abnormal. The pulse tracing corresponds.

MITRAL OBSTRUCTION: MITRAL STENOSIS.—This lesion is believed to be usually of rheumatic origin, but very often the connection cannot be traced, and the cause remains altogether obscure. It is more common in young women. The pathological characters of obstructive lesions are described at p. 330. These are developed in great variety in connection with the mitral orifice and valvular

apparatus, and may be said to belong to two principal types. The first of these is the vegetative, in which the obstruction mainly occupies the auricular surface of the mitral cusps, and consists of irregular masses of growth and organised or calcified thrombi projecting into the ostium (see Fig. 24, p. 328). The second type is the sclerotic. In this the morbid process involves the auriculo-ventricular ring proper, which gradually undergoes stricture; or the chordæ tendineæ and margins of the segments are so thickened or fused together that the mitral orifice is represented by a buttonhole-like chink, or a small aperture at the extremity of a funnel-shaped structure. The two types may be combined. Compensation is effected mainly by hypertrophy of the right ventricle—which becomes gradually dilated as failure occurs, and also by similar changes in the left auricle; the left ventricle is small as the result of diminished intake and output. The pulmonic circulation, venous system, and viscera are disturbed in the same way as in mitral incompetence, and the usual changes in these, as well as dropsy, attend cardiac failure.

The **symptoms** of mitral obstruction closely resemble those of incompetence; but the patient is more often a woman, of short stature and small build; the cheeks present a brighter or less livid flush; and bronchial and pulmonary catarrhs, with occasional attacks of transient pulmonary congestion of an acute type accompanied by urgent dyspnoea and hæmoptysis, are more common. Bronchitis in the colder seasons, and other causes of pulmonary stress, and in women pregnancy and parturition, readily lead to cardiac failure, which follows its usual course. The disease is not incompatible with long life; but as a simple result of endocarditis with repair it is peculiar amongst valvular lesions in occasionally being progressive like other fibroid strictures, and cardiac failure from this cause is essentially hopeless.

With the exception of the præcordial dulness, which affords the usual evidence of moderate enlargement of the right side of the heart, the **physical signs** are strikingly characteristic as long as compensation lasts. They include thrill in the mitral area, which is usually presystolic, but sometimes occupies most of the diastolic time, followed by a sudden, short, thumping impulse; presystolic murmur (p. 351); a sudden, smacking, abrupt first sound, which is commonly audible with the unaided ear some distance from the chest; and a reduplicated second sound, which is accentuated in the pulmonic area. All these phenomena vary in different cases, and in the same case under different circumstances.

Thus in quiet recumbency the frequency of the heart falls and the murmur may disappear; to return when the heart is excited by rising, or by raising the arms. When the heart fails, the thrill and murmur may disappear, the first sound become weak and irregular, and a systolic murmur develop in the tricuspid area; the original signs returning along with compensation. In some instances of mitral stenosis neither thrill nor murmur is present; but the peculiar first and second sounds are rarely absent, and may be recognised over the whole præcordia and even over the back. Thus the physical signs of this lesion are the most uncertain and variable of clinical phenomena in connection with valvular disease. The pulse is small, and of various tension and force; it is by no means irregular as it is in mitral incompetence, provided the lesion be compensated. The sphygmogram presents corresponding characters.

AORTIC OBSTRUCTION.—Obstruction at the aortic orifice is less often of rheumatic origin than a result of chronic strain, arterial degeneration, and syphilis. For this reason the male sex, middle and advanced age, and laborious occupations are etiologically related to it.

The anatomical characters of obstructive valvular lesions have been described at p. 330. At the aortic orifice they sometimes take the form of vegetative masses, of great variety in size and shape, the result of endocarditis and associated processes (including thrombosis and calcification), which project into the lumen, either above or below the level of the free borders of the cusps. More frequently the valves are found thickened, irregular, shrunk, everted, or mutually adherent (see Fig. 23, p. 328). The left ventricle is the seat of pure hypertrophy until failure occurs, when it becomes dilated also, and the change may progress backwards as usual through the different chambers of the heart, usually with consecutive mitral incompetence. The root of the aorta and the mouths of the coronary arteries are liable to be involved, with serious consequences to the nutrition of the myocardium, particularly in syphilis, atheroma, and acute injury.

In aortic obstruction with full compensatory hypertrophy there are theoretically no morbid **symptoms**, but any increased demand on the heart may produce faintness and giddiness from cerebral anæmia, whilst præcordial discomfort, palpitation, and pain also occur very readily in connection with flatulence. When compensation breaks down, the usual symptoms of cardiac failure are added to these. This happens most often in consequence of vascular degeneration (gout, syphilis, Bright's disease, etc.), poverty, and

continued strain; but notwithstanding the progressive character of some of these conditions, compensation may be temporarily regained. Sudden death is not uncommon.

The **physical signs** in compensated cases are as follows:—The apex-beat is definite and localised at a spot downwards and outwards from the normal in the fifth or sixth interspace, and the area of præcordial dulness is increased in the same direction. Systolic thrill may be present at the aortic area, and here and over the manubrium an area of dulness can often be made out, significant of associated dilatation of the aorta. The characteristic auscultatory sign is the aortic systolic murmur (p. 348). The first sound is usually inaudible therewith; the second aortic sound may be loud and ringing from disease of the arch. The pulse-wave is regular (or irregular, particularly in senile cases), slow, sustained, delayed; the vessel-walls are of various size and tension, and often degenerated, according to the associated conditions. The sphygmogram presents low altitude, a rounded summit, an ill-developed dirotic wave, and increased duration. When compensation is undone, the ordinary signs of failure of the heart are developed, including a mitral systolic murmur from leakage due to dilatation of the left ventricle, and the pulse alters accordingly.

AORTIC INCOMPETENCE.—This important form of valvular disease originates in the same causes as aortic obstruction, which often accompanies it. Strain, syphilis, and acute endocarditis are, however, more common causes of it than is simple atheroma.

The **morbid anatomy** of incompetent lesions is described at p. 330. As they involve the aortic valves, they may be said to be identical with those which give rise to obstruction in the same situation, the two forms of valvular disease at this orifice being commonly associated. This description applies also to the aorta and the coronary arteries. In some instances one or more of the cusps are torn, as the result of acute strain; in others a perforation in a single cusp permits regurgitation. The aortic arch is dilated and may be extensively diseased. The left ventricle is primarily dilated from being overfilled, and is consequently hypertrophied; the enlargement may be very great. After death from failure of compensation, further dilatation is found, due to insufficient emptying, and may involve both sides of the heart, the process having been developed backwards as usual. The whole arterial system is dilated, the smaller vessels being visibly lengthened and tortuous, whilst their walls are thickened.

The special **symptoms** of aortic incompetence are mainly

visible anæmia, dyspncea, giddiness, and occasional faintness or actual syncope, due to the discontinuous and insufficient flow and pressure of blood in the systemic vessels; and pain, which often amounts to angina pectoris, and attacks of palpitation with præcordial distress and anxiety, caused by engorgement of the left ventricle and aorta. But compensation is ordinarily sufficient and the patient free from cardiac symptoms of every kind. If the heart fail, the familiar clinical phenomena of this state are super-added, the mitral valve sometimes becoming incompetent. Death may occur in this way, or suddenly from syncope, or in angina. The course of aortic incompetence is usually shorter and more uncertain than that of other forms of chronic valvular disease, both from its own characters and effects and because of its nature. Indeed, syphilitic disease in this situation may be subacute in its course; and rupture of the aortic valves by strain may prove fatal rapidly. Aortic incompetence is a common cause of sudden death.

The **physical signs** include those of dilatation and hypertrophy of the left ventricle, with increased cardiac action, the impulse being heaving, the apex-beat displaced far down and to the left, and the præcordial dulness correspondingly increased; aortic diastolic murmur, with or without audible second sound, in the aortic area and in cervical vessels; occasionally diastolic thrill, basic or apical; weak visible impulse in the aortic area, and throbbing of the cervical vessels. To these, other signs are added if the heart fail. The characteristic pulse is long, thick, tortuous, locomotive, and very soft; the pulse-wave is sudden, quick, very ample, strong, and regular. The tracing exhibits great altitude of the primary wave with sudden descent and ill-marked dicrotic wave. Capillary pulse is found in the usual situations; rarely a true venous pulse also. In some instances a presystolic murmur is audible in the mitral area, less loud and less definite than that characteristic of mitral stenosis, and even more variable than it. This physical sign, commonly known as Flint's murmur, appears to be due to interference with the anterior mitral cusp, and so with auricular output, by the current of blood regurgitating through the aortic opening. Duroziez's murmur may be found in the arteries (p. 356).

TRICUSPID INCOMPETENCE.—Incompetence of the right auriculo-ventricular valve is found under two different circumstances, namely, (1) (rarely) as a primary lesion of the cusps, which is usually rheumatic; and (2) (commonly) as a secondary dilatation of the opening in association with dilatation of the right ventricle from any cause, particularly mitral and chronic pulmonary diseases,

as well as in debility. The right ventricle is dilated and hypertrophied; the right auricle is dilated and hypertrophied; the left chambers are frequently the seat of valvular lesions and enlargement. Therewith the ordinary evidences of cardiac failure are found in the veins, viscera, serous sacs, and subcutaneous tissues. The **symptoms** of tricuspid incompetence are practically those of cardiac failure. The characteristic **physical signs** are the tricuspid systolic murmur (p. 350), venous pulsation in the neck, and pulsation of the liver, along with the other phenomena of dilatation with failure.

Secondary tricuspid incompetence is often but temporary, passing off with restoration of compensation of the right ventricle; but it usually returns with subsequent failure, which sooner or later proves fatal.

TRICUSPID OBSTRUCTION.—This is a comparatively rare lesion. Its etiological relations are the same as those of mitral obstruction, with which it is commonly associated, and which it very closely resembles in its symptoms and course. Its characteristic *physical sign* is the tricuspid presystolic murmur, which may however be wanting.

PULMONIC OBSTRUCTION AND PULMONIC INCOMPETENCE.—These are the rarest forms of acquired valvular disease. They are rheumatic, septic, or traumatic in origin. Their pathological characters closely resemble those of aortic lesions. In some instances the conus is sclerosed and stenosed. The right ventricle is hypertrophied in obstruction, dilated and hypertrophied in incompetence. These lesions are characterised clinically by dyspnœa, by præcordial distress and anginal pain, which may extend down the right arm, and by the pulmonic systolic and diastolic murmurs, with the ordinary physical signs of enlargement of the right ventricle. A weak diastolic pulmonic murmur, which may occur temporarily without valvular lesion, is believed to be significant of functional incompetence, the result of high pulmonic pressure.

COMPLEX VALVULAR DISEASE: ASSOCIATED LESIONS.—In a large number of instances chronic valvular disease is not a simple lesion or confined to one set of valves. Aortic obstruction usually accompanies aortic incompetence; mitral disease is frequently double; aortic incompetence and mitral obstruction are occasionally associated; and we have seen how mitral and tricuspid incompetence are natural sequelæ of other lesions, respectively, which dilate the ventricles. The symptoms, signs, and course of complex valvular disease present the characters of the different forms variously

blended, and their termination is usually that which is common to all, namely, cardiac failure, with dropsy.

Diagnosis.—Chronic valvular disease is simulated by various morbid conditions. (1) It must not be confounded with functional disorders of the heart, particularly those associated with hæmic murmurs (p. 433), but the two are often combined. (2) Acute endocarditis is differentiated as described on p. 396. (3) Temporary leakage of the mitral or tricuspid valves in cardiac failure consequent on chronic nephritis, etc., is distinguished by the proper phenomena and course of such affections; and *vice versa* it is simulated by valvular disease with failure when the murmur has temporarily disappeared. (4) Aneurysms and other diseases in the chest, such as malignant disease, accompanied by murmurs, require nothing more than ordinary clinical care for their discrimination (pp. 454 and 280). (5) Mitral systolic murmur is simulated by the cardio-pulmonary murmur, audible in or to the left of the mitral area, and due to movement of air in the lung by excited action of the heart during physical examination. Further, it must never be forgotten that the complete diagnosis of every case of valvular disease should include (*a*) the *form* of lesion, which is determined mainly by physical examination; (*b*) the *nature* or kind of lesion, ascertained by investigation of the cause; (*c*) the existence of *compensation* or of *failure*, with their respective symptoms and signs; and (*d*), in failure, the *cause of failure*, elicited by attentive inquiry into the patient's circumstances and recent history. Nothing less than this knowledge suffices for rational prognosis and treatment.

Prognosis.—The prognosis of chronic valvular disease is based upon many considerations, which are analysed and fully set forth on pp. 381 *et seq.* Briefly, when the lesion is compensated these are as follows:—(*a*) As regards the *form* of lesion, the order of gravity is aortic incompetence, mitral stenosis, mitral incompetence, aortic stenosis. (*b*) The *origin* or *kind* of disease is of paramount importance prognostically, degenerative being far more serious than old rheumatic lesions, and in this connection the associated conditions of other viscera, such as the kidneys, as well as of the vessels, must always be considered, which may prove disabling or fatal otherwise than through the heart. (*c*) The *severity* of the lesion—the degree of stenosis or incompetence respectively, and the number of valves involved—will always be reckoned with. (*d*) Lastly, prognosis is vain without a knowledge of the *security of compensation*, otherwise the risk of failure, which varies widely with

age (each period of life presenting its own dangers—fresh rheumatism, syphilis, alcoholism, gout, senility, respectively), with sex (women incurring a number of special dangers), and with social position, occupation, habits, temperament, etc., as already described.

When compensation fails the prognosis must be estimated by the first three considerations above ; by the cause of failure, whether this be removable temporarily, like overwork and under-feeding, or irremovable, like increase of the lesion, or uncertain, like many nervous causes ; and by the urgency of the clinical phenomena (p. 387).

Treatment.—The rational treatment of valvular disease of the heart is (1) preventive, and (2) remedial.

1. *Preventive treatment.*—A large proportion of all cases of valvular disease commence with acute endocarditis ; and the control of this disease, and the judicious management of it by prompt, thorough, and prolonged attention, including protracted rest of body and mind during convalescence, are the most promising of all prophylactic measures (p. 389). Further, as rheumatism, the chief cause of endocarditis, is essentially recurrent, there is constant opportunity to prevent valvulitis and pericarditis, and the increase of chronic valvular and pericardial lesions. The second great class of morbid conditions of the valves are degenerative, often directly or indirectly referable to unwise, unwholesome, or unfortunate modes of living. These might be prevented by greater respect for hygiene generally than most persons exercise in everyday life : by careful diet ; moderation in the use of stimulants and other luxuries ; regulation of physical and mental work, relaxation and exercise ; and attention to the elementary functions of the body, particularly elimination.

2. *Remedial treatment.*—When prevention has failed, and chronic valvular disease is established, treatment follows the principles laid down in a previous section (p. 390).

In the phase of *compensation*, which it is our primary object to maintain, the patient's occupation has to be discussed, and may have to be directed and controlled, including his work, rest, and exercise, and in the young subject his education. To do so an estimate is first made of the capacity of his heart for work, based on the nature, form, and extent of the valvular lesion in relation to his age, his circumstances, the state of his other organs, and his constitution as a whole. The bearing of a correct prognosis on the management of compensation is now fully appreciated. Definite rules are laid down for each patient. In the case of *children and*

youths disabled by valvular disease, these mainly relate to their education and physical training. The various studies and games which may be permitted are individually considered, regard being had to the injurious effects of intellectual and muscular stress on the one hand, and of mental and bodily torpidity on the other. At the same time every effort must be made to prevent the return of endocarditis and fresh lesions. *During manhood and early middle age* the subject of compensated valvular disease has the management of his case in his own hands. This will consist in strict moderation of work whether physical or intellectual, of exercise, of amusements, and of indulgence in luxuries generally, including alcohol and tobacco. He often finds this a difficult matter, as the duties and pleasures of domestic, social, and public life offer constant temptation to the adult subject of compensated valvular disease to forget his disability, and to indulge his ambitions or his love of sport instead of keeping the calls on his circulation down to the level of his damaged heart. Periodical examination of the heart and vessels, along with a review of the patient's circumstances, is a practice to be observed. *In later middle life and old age* the treatment of compensated valvular disease principally aims at preventing or retarding degeneration of the coronary arteries and myocardium. This object is most likely to be effected by insuring temperance in eating and in the use of stimulants, strict attention to elimination by skin and bowels, and regular moderate physical exercise, whether active or passive, in fresh air, as distinguished from intermittent resort to unwise exertion at long intervals. Atheroma connected with gout, glycosuria, or obesity might be arrested by such reform of diet and habits generally, coupled with the special medicinal treatment indicated in these disorders severally. Degenerative valvular disease in middle-aged and older individuals, whose manner of living presents no discoverable cause for nutritional failure beyond anxiety and worry, will suggest temporary, or it may be permanent, relief from business or other harassing occupation. If the morbid changes in the valves, myocardium, and arteries are progressive, they may sometimes be arrested by these measures; but it is always well to take advantage of the additional help of iron to improve the blood, of arsenic to modify the local metabolism, of cardiac tonics, like digitalis and strychnine in small doses, and especially of well-planned exercise, movements, and baths, to invigorate and rehabilitate the muscular tissue. Chronic strain of the valves is itself irremediable, but obviously requires to be treated with comparative rest.

At all ages the influence of season and weather in this country on compensated valvular disease must not be forgotten. In mitral and tricuspid lesions, particularly if they be severe, advanced, or already complicated with bronchial and pulmonary changes, the colder and more unsettled months of the year should be spent in a mild climate; and if this cannot be done, the patient's clothing and the temperature and ventilation of his rooms must be supervised.

Compensation in valvular disease is promoted by occasional resort to drugs. In younger subjects hæmatinics are useful, such as the different preparations of iron and arsenic; with these strychnine may be combined as a cardiac tonic, and occasionally small doses of digitalis, for example:—

R. Tincturæ ferri perchloridi, ℥x.
 Tincturæ digitalis, ℥v.
 Liquoris strychninæ hydrochloridi, ℥iij.
 Acidi phosphorici diluti, ℥xv.
 Aquæ ad ℥j.
 Three times a day after meals.

A syphilitic lesion indicates the employment of iodides or mercurials, and at the same time both physical and mental rest, because of its relatively acute progress, and the probable involvement of the coronary arteries and myocardium in the specific process, along with the valves.

The disturbances and embarrassments of the compensated heart which will occur from time to time, particularly in older subjects, rarely require direct cardiac treatment. Careful diet, purgatives, hepatic stimulants, and stomachics are usually more appropriate and successful remedies, along with such a reform of living as has already been described. But immediate relief ought to be afforded with carminatives and diffusible stimulants, such as spirit of ether, aromatic spirit of ammonia, compound tincture of cardamoms, and ginger.

Failure.—The indications for the treatment of cardiac failure in general have been already discussed at p. 391. The order in which these should be severally attended to in practice, when failure occurs in connection with chronic valvular disease, is determined mainly by the urgency of the patient's condition. First, the work of the heart is reduced by securing bodily rest. The patient is confined to bed, his back well supported with high pillows; or he occupies a large easy-chair in a well-ventilated room, with light, loose, but warm garments. He is spared every kind of

excitement ; anxiety is allayed by the cheerful reassurance of those around him ; and the most urgent forms of distress are relieved by measures presently to be described under "complications." In thus affording rest and comfort of body and mind, we remove two of the commonest causes of failure—the physical stress of effort on the systemic circulation and left ventricle, and the respiratory stress produced by bronchial catarrh on the pulmonic circulation and right ventricle. Secondly, venous engorgement and the burthen of arrears of cardiac work may have to be reduced immediately in the most urgent cases by the abstraction of blood with leeches or cupping over the præcordia, or by venesection to five or ten fluid ounces. Far more commonly it is sufficient to obtain repeated hydragogue motions, by means of compound jalap powder (30 to 60 grains), or with a combination of colocynth and hyoscyamus and blue pill (5 to 8 grains) followed by magnesium or sodium sulphate, or with compound elaterin powder (3 to 5 grains).

It now remains to plan the measures intended to fulfil the third leading indication for the treatment of cardiac failure, namely, to increase the force of the heart, and restore the effectiveness of systolic output. In a number of instances this can be done by attention to diet alone. The food must always be definitely ordered, not left to the judgment of the patient's friends or a nurse, who are disposed to give him frequent feeds of slops, with the effect of producing gastric distension, flatulence, and epigastric or præcordial oppression. The proper diet in cardiac failure, as described in detail at p. 393, consists chiefly of very digestible, nutritious solids, given at regular meal-times only ; and these the patient himself will be found to prefer. If they cannot be taken or retained at first, or if the stomach become disordered by irritant drugs, such as digitalis, solid food is replaced for a day or two by peptonised milk, beef-tea, or the like, in small quantities, as will be described presently in connection with the management of vomiting (p. 413). A few fluid drachms of brandy or whisky in an aerated water, or two ounces of sound red wine, are usually ordered with the mid-day meal. Correct dietetic treatment has the further advantage that it strikes at some of the commonest causes of cardiac failure that may have been at work, namely, insufficient or injudicious nourishment, dyspepsia, anæmia, chronic alcoholism, gout, glycosuria, Bright's disease, or associated diseases of digestion and assimilation, and thus may be sufficient to restore compensation. Purgatives contribute to this end. It is usual, however, to reinforce the influence of food on the failing heart with drugs that directly increase the vigour of

ventricular contraction, and otherwise aid the circulation. Digitalis, strophanthus, squill, senega, convallaria, or allied drugs that favourably influence the heart and vessels and promote diuresis, are prescribed at once; but as they do not sensibly increase the amount of urine (the best test of their action) for at least three days, it is often necessary meanwhile to administer strychnine subcutaneously (gr. $\frac{1}{30}$ - $\frac{1}{20}$), or by the mouth (5 minims of the solution of the hydrochloride in combination with the other drugs), several times a day, as well as ether and ammonia draughts, as may be demanded by dyspnœa, faintness, or præcordial distress, in order to tide over this period. Digitalis is ordered along with diuretics of other kinds, for example:

R Potassii acetatis, gr. xx.
Tincturæ digitalis, ℥x.
Tincturæ scillæ, ℥xx.
Liquoris strychninæ hydrochloridi, ℥iv.
Infusi senegæ, ad ℥j.
Every four hours.

Or,

R Digitalis foliorum, gr. j.
Scillæ, gr. j.
Pilulæ hydrargyri, gr. j.
Three times a day after meals.

Care must be taken to give full amounts of these drugs; and continued severity of cardiac symptoms is more commonly due to insufficient than to excessive dosage. With free diuresis marked relief of distress commonly occurs; and this can be maintained by continuing, in gradually diminished amounts and otherwise modified, the various measures that have been employed.

A number of *complications* or urgent developments demand special consideration in the treatment of cardiac failure, such as dyspnœa, præcordial oppression, palpitation, faintness, pain, vomiting, and insomnia. These variously depend on the disturbances of circulation and their results in different organs, particularly the stomach and bowels, and they yield naturally to radical treatment, that is, to treatment directed to the failing heart, improvement in all respects setting in with rest and reduction of venous engorgement, and advancing rapidly as diuresis is re-established. At the commencement of most cases, however, and in cases that do not yield to radical treatment, these different forms of distress also demand direct and immediate relief.

Whilst continuous *dyspnœa* disappears under the influence of

rest (particularly in a chair), purgation, cardiac stimulation, and, if necessary, paracentesis of the thorax or abdomen, fits of urgent breathlessness have to be relieved with ether, ammonia, or trinitrin. The same remedies are used in *præcordial oppression*. This also yields, when it is caused by flatulent dyspepsia, to simple carminative and antacid drugs, such as cardamoms, ginger, aromatic spirit of ammonia, and sodium bicarbonate in suitable combinations. When it is severe, amounting to anguish, a hypodermic injection of gr. $\frac{1}{8}$ of morphine acetate, combined with gr. $\frac{1}{30}$ of strychnine hydrochloride, or the application of a few leeches over the præcordia, affords most speedy relief. The most readily available treatment for *faintness* is brandy in doses of 2 to 4 fluid drachms in water, or the ether and ammonia combination, but strychnine or ether subcutaneously may be necessary, or either brandy or ether per rectum. Fresh air, fanning, and warm applications or friction to the extremities are methods always usefully employed. Moderate *pain* can usually be relieved with warm applications, either dry or moist, to the præcordia. When acute it will not yield to less powerful remedies than morphine (in the above form) or the nitrites, which are particularly effective in angina pectoris—one tablet of trinitrin to be hastily chewed, or four minims of amyl nitrite inhaled from a crushed capsule. *Palpitation*, as it occurs in fits in the course of cardiac failure, ought to be prevented by strict control of diet and by avoidance of excitement, and unwise or unnecessary movement. When caused by flatulence it is best arrested by raising the patient carefully into the sitting posture, with the legs dependent, and the use of one or other of the stimulant, antispasmodic, or carminative combinations already mentioned, to produce eructation. *Vomiting* is always a disturbing and sometimes a grave complication of cardiac failure, as it interferes with the administration of food, and, being aggravated, if not actually caused, by digitalis or other necessary drugs, compels their intermission. It is best met with very spare fluid food, such as peptonised milk or meat essences, or even brandy-and-water only; with calomel, followed by a saline purgative; and with a few doses of bismuth subnitrate, sodium bicarbonate, sal-volatile, and diluted prussic acid. When the stomach has become quiet for a few hours, light solids are again tried; and if these are retained, digitalis is cautiously resumed. Should vomiting return, rectal alimentation and the hypodermic use of strychnine or digitalin, or both, possibly with morphine, must be resorted to. *Insomnia* is not to be treated in a routine fashion, but if sleep fail in spite of radical treatment, sulphonal, chloralamide, or paraldehyde is given with great advantage;

for continued insomnia, particularly in association with præcordial anguish, dyspnœa, and restlessness, speedily exhausts the heart and seriously reduces the prospect of recovery. In the worst cases hypodermic injections of gr. $\frac{1}{12}$ or gr. $\frac{1}{10}$ of morphine, with gr. $\frac{1}{25}$ of strychnine, are employed with great advantage.

The treatment of *dropsy* is described later.

The success of the treatment employed is estimated during the first two or three days by the relief of distress and general urgency of the patient's condition, then by the decline of dropsy, and about the third or fourth day by striking and possibly sudden diuresis, the volume of urine mounting from perhaps 20 to 60 or even 100 fluid ounces. Therewith the characters of the pulse and physical phenomena over the heart gradually become significant of increased cardiac force and diminished cardiac dilatation. The treatment is now modified. The frequency of purgation and the doses of the different drugs are reduced. Palliative remedies, such as carminatives and hypnotics, may be no longer required. The diet is made somewhat more generous in amount. Presently a little pleasing mental occupation is allowed, but bodily rest is continued. As the amount of digitalis is further reduced, iron is cautiously combined with it;

R Digitalis foliorum, gr. j.
 Ferri sulphatis, gr. j.
 Quininae sulphatis, gr. j.
 Piperis nigri, gr. ss.

Three times a day immediately after meals.

Thus the case progresses towards the re-establishment of compensation; and now the patient is permitted to move about the room, the effect of this step upon his breathing and pulse, and the possible return of œdema of the ankles, being closely observed. As a rule it is many weeks, and it may be many months, before he is able to resume his occupation; and before and after doing so he should observe the rules for the establishment and maintenance of compensation which have been laid down above.

Want of success at the commencement of treatment of cardiac failure causes disappointment, but never despair. After all the measures enumerated have proved insufficient, and, in particular, when dropsy refuses to yield to them, puncture of the legs or paracentesis abdominis, under rigidly antiseptic conditions, often turns the scale in a favourable direction. All the other remedies are diligently employed at the same time, and, as has been already

said, their failure suggests increased doses, 15 minims of tincture of digitalis being given every four hours, an occasional draught of 5 to 8 grains of caffeine citrate combined with 10 grains of sodium salicylate to induce rapid diuresis, and strychnine hypodermically three times a day.

Two other subjects connected with the treatment of valvular disease remain for consideration—the *nature* of the lesion and the *form* of the lesion.

When the valvular disease is not inflammatory but degenerative in its *nature*, an attempt must be made to deal with the particular cause that is at work by means of appropriate measures, medicinal and non-medicinal, the importance of nutrition in the complete sense of the term being steadily kept in view.

The *form* of the valvular lesion—whether mitral or aortic, obstructive or regurgitant—is, speaking broadly to begin with, of much less moment therapeutically than the stage of the disease (that is, the condition of the myocardium) and its pathological nature. Still, it is necessary to remember, in the management of compensated valvular disease, that the subjects of mitral lesions must be specially guarded against respiratory affections, and, in the case of women, cautioned against pregnancy; whilst strain and the common causes of anæmia must be more diligently avoided in aortic lesions. The same considerations demand attention in the treatment of failure. The suggestion that digitalis is less suitable in aortic incompetence because it lengthens diastole, and thus increases the period of overfilling of the left ventricle, may be disregarded practically. Not overfilling, but insufficient emptying, is the dynamic disturbance that gives concern and demands treatment in failing heart.

DISEASES OF THE MYOCARDIUM

ACUTE MYOCARDITIS

Like the endocardium and pericardium, the muscular walls of the heart usually become inflamed under the influence of rheumatism or one of the specific contagia, particularly diphtheria, scarlet fever, and septicæmia. The pathological changes are described at p. 326. Septic myocarditis may lead to abscess and acute cardiac aneurysm; the other kinds rarely end in visible suppuration, but occasionally in fibrosis.

The **clinical phenomena** of acute myocarditis make their appearance in the course of rheumatism or of one of the acute infective diseases of a severe type, of which it is but a part; and they are very rarely to be distinguished from the symptoms of cardiac excitement ending in failure and the disturbances of the pulse that characterise fevers. When fully developed, they include præcordial pain and distress, palpitation, dyspnœa, lividity, restlessness and anxiety, along with the ordinary physical signs of dilatation and great weakness of the heart, and unnatural feebleness, infrequency, acceleration, or irregularity of the pulse. Signs of associated endocarditis and pericarditis increase the difficulty of the diagnosis, but at the same time increase the probability of the existence of myocarditis. Excepting in rheumatic cases, the course of acute myocarditis is rapid, for example in diphtheria; and the usual result is death, which may be sudden.

The **prognosis** is therefore very grave. So seldom is this disease diagnosed with certainty that the **treatment** followed in any case cannot be said to be different from that of the primary disease and the accompanying inflammation of the endocardium and pericardium. Specific remedies directed to the cause, such as salicylates or antitoxin, skilled nursing, absolute bodily and mental rest, local sedatives, careful nutrition with predigested food in small quantities at short intervals, and cautious stimulation of the heart with strychnine and alcohol, are all obviously indicated. If acute dilatation set in, the proper remedies must be employed, including abstraction of blood and the administration of oxygen. Prolonged rest during convalescence is as necessary as it is in other forms of acute cardiac disease (p. 389).

CHRONIC MYOCARDITIS—FIBROID DISEASE

The nature, origin, pathological characters and course, and various relations and terminations of these lesions of the cardiac walls have been discussed in connection with "Disorders and Diseases of Nutrition—Fibrosis" (pp. 324 *et seq.*).

The **clinical phenomena and course** of chronic myocarditis and its effects are so intimately associated with those of arterial degeneration, and of cardiac failure in valvular disease or chronic nephritis, that they cannot be described as distinctive, nor can the parietal disease be *diagnosed* with any certainty. The prominent features are those of chronic cardiac weakness, particularly feebleness, infrequency, and irregularity of the pulse, angina, dyspnœa, and

faintness. Life is insecure, and death may occur suddenly and unexpectedly. **Treatment** should be directed to meet the cause, to strengthen the myocardium, and to prevent functional disturbances of the heart calculated to be distressing and dangerous, particularly by attention to digestion, regulation of the bowels, and avoidance of exertion and excitement.

CARDIAC ANEURYSM

The modes of origin of aneurysm of the cardiac walls have been described in connection with the pathology of acute and chronic myocarditis, fibroid disease, and syphilis (pp. 326 and 327). Clinically the disease is rarely differentiated from valvular lesions, both the symptoms and the signs being very similar in the two affections. Anomalous bulgings and dull percussion areas over the præcordia, and murmurs of peculiar characters and distribution, might suggest the existence of this rare condition. The prognosis is bad. Treatment would have to be directed to cardiac degeneration.

FATTY DEGENERATION

The causes of fatty degeneration of the myocardium and its *post-mortem characters* constitute one of the most important subjects of the general etiology and pathology of diseases of the heart (see pp. 318 and 323). It commonly originates in local anæmia from coronary disease; less frequently in general anæmia, the acute infective fevers, and chronic alcoholism.

The **symptoms and signs** of fatty degeneration are those of chronic feebleness and disorder of the cardiac action—whether acceleration, infrequency, or irregularity of rhythm—and disturbance of cardiac sensibility. More characteristic is the occurrence of seizures of acute cardiac failure at irregular intervals and under the influence of exertion, excitement, general anæsthetics, or flatulent distension: faintness or actual syncope, præcordial anxiety, arrest of movement and of respiration, possibly angina pectoris, and sometimes coma with imperceptible pulse. The result may be sudden death; and the gravity of the condition is evidenced by the increasing readiness with which the seizures are induced by trifling causes.

The **diagnosis** turns on the existence of such symptoms independently of valvular disease; and it is to be understood that chronic gout, arterial sclerosis, Bright's disease, or syphilis, singly or combined, is often associated with the myocardial degeneration.

An acute variety of the disease is one of the elements of profound anæmia from hæmorrhage. The diagnosis is extremely difficult to determine absolutely. The **prognosis** is very grave.

Treatment has to be directed, first, to the primary pathological changes in which the degeneration has originated. These will demand thorough consideration of the patient's mode of living, particularly in respect of diet and exercise. Graduated movements and baths are sometimes of real value in this disease. In the second place, the circumstances which might excite an anginal seizure must be controlled. The most useful drugs in fatty degeneration of the heart include stomachics, laxatives, cardiac tonics, arsenic, iodides, and hæmatinics given sparingly and cautiously. Alcohol, ether, ammonia, strychnine, and particularly the nitrites are of great value in the acute attacks.

FATTY INFILTRATION—COR ADIPOSUM

The existence of fat-laden heart, the pathology of which is described on p. 324, may be diagnosed when obese persons, who are habitually scant of breath, also present the subjective and objective symptoms and the physical signs of chronic cardiac weakness, such as faintness, an irregular feeble pulse, and nearly inaudible cardiac sounds, and suffer from attacks of cardiac distress in connection with indigestion. The course, diagnosis, prognosis, and treatment of this morbid state are all essentially dependent on the other elements of the adipose habit, particularly glycosuria, gout, and chronic nephritis. In planning the dietetic management of the case, care must be taken to prevent the occurrence of flatulent distension.

CARDIAC SYPHILIS

Syphilitic gummata of the myocardium, and arteritis involving the coronary vessels, as well as the nutritional changes in the walls of the heart which result from them, are described on pp. 325 and 335. The disease is not common, and is seldom differentiated from other kinds of myocardial degeneration. Highly suggestive features include disturbances of the rate and rhythm of the heart and pulse; the appearance of cardiac degeneration at a relatively early age; the history and present evidences of specific disease; and the success of treatment directed to it; or on the contrary the occurrence of sudden death in such a subject free

from valvular disease. The prognosis and treatment are obvious.

ENLARGEMENT OF THE HEART

DILATATION AND HYPERTROPHY

Etiology.—Valvular disease is the principal but by no means the only cause of disturbance of the circulation that gives rise to enlargement of the heart. A number of other morbid influences tax the left or the right ventricle, or the heart as a whole, induce hypertrophy or dilatation, and lead to cardiac failure at last if the patient have not died earlier from some associated disease. Independently of disease of the aortic and mitral valves, enlargement of the *left* ventricle is produced by high arterial tension; arterial sclerosis or atheroma, or their causes, such as nephritis, gout, alcohol, syphilis, chronic muscular stress, and senility; also by coarctation of the aorta. Enlargement of the *right* ventricle, besides its association with disease of the mitral and pulmonic valves, is consequent on pulmonary emphysema and fibrosis, pleuritic adhesion and chronic bronchitis, and is also the permanent result of acute over-distension with cardiac strain during severe muscular efforts. Amongst the causes of *general* cardiac enlargement are persistent or chronic over-action of the heart, whether from physical or mental causes, as in laborious occupations and Graves' disease, pregnancy and plethora, chronic myocarditis and myocardial degenerations, and adherent pericardium.

Anatomical characters.—These naturally vary with the cause of the enlargement. In the simplest and most instructive instances of all, aortic obstruction and chronic Bright's disease with high arterial tension, the form of enlargement of the left ventricle is at first pure hypertrophy. Theoretically a similar condition of the right ventricle exists in the early stage of mitral lesions and pulmonary emphysema. In most of its other associations, cardiac enlargement is partly dilatation—dilatation from over-filling of the left ventricle in aortic incompetence and in mitral incompetence; dilatation from over-filling of the right ventricle consequent on severe exertion (including that of prolonged or repeated dyspnoæal effort and cough) or laborious occupations, and on pulmonic or tricuspid incompetence; residual dilatation of either ventricle from failure of the heart, the result of impaired nutrition of the myo-

cardium; and the latter is the final result in every case, sooner or later. The structural changes in hypertrophy and dilatation are described at p. 331. The histological characters of the cardiac walls vary widely: either normal, fatty, or fibroid otherwise degenerated tissues are found in different instances and at different stages. Even when they are not primarily diseased, the valves are often opaque and thickened, and the auriculo-ventricular openings secondarily dilated. The other viscera present every variety of pathological change. Some of these, like chronic nephritis, are causally associated with the cardiac enlargement; some are common effects with it of the morbid cause—for example, alcoholic hepatitis; and others are the results of failure of the heart, particularly mechanical congestion.

Clinical characters.—The leading symptoms and signs of this condition are those of the primary disease on which cardiac enlargement has supervened—valvular disease, emphysema, nephritis, etc. In connection with the first of these they have been already fully discussed; apart from them they do not call for lengthened description.

The **symptoms** of *simple hypertrophy* of the heart, considered theoretically, are, perfect health and disappearance of the circulatory disorder and distress produced by the associated morbid conditions just enumerated. With respect to the heart itself, the clinical phenomena are at first symptomatic and significant of sufficiency and vigour: the condition is one of compensation or the absence of disability. The **physical signs** of simple hypertrophy are different according to the chamber or chambers enlarged, and they have therefore to be described broadly. The præcordia may be prominent, particularly in young subjects. The apex-beat is definite, localised, and thrusting. Increase of the præcordial dulness in the direction of growth is chiefly downwards at the apex. The first sound becomes more toneless, or dull and sustained; it is sometimes nearly or quite inaudible at the aortic area. The second is often accentuated at either base. But these characters of the sounds, and the different murmurs which are so often present, vary with the primary morbid state.

The symptoms of *dilatation from over-filling in association with hypertrophy* are also theoretically negative; the clinical phenomena of the primary lesion are prevented or removed by the oncome of the enlargement, that is, compensation is established. However, the condition is more precarious than in simple hypertrophy. The limits of the capacity of the heart to meet either

physical or mental stress are narrower ; and temporary attacks of cardiac or respiratory embarrassment not infrequently occur. The physical signs are those of increased size and vigour of the heart—præcordial bulging, heaving impulse, and displaced apex-beat downwards and outwards ; corresponding increase of the præcordial dulness in all directions, especially transversely towards the left ; and, as a rule, a murmur due to valvular disease.

Course.—The course of cardiac enlargement when it is secondary to valvular disease is described at p. 398, and that of compensatory enlargement in general and its termination in failure at p. 378. When it is compensatory to other than valvular lesions, enlargement is usually more precarious. The primary disease is not only permanent but often progressive, like nephritis and emphysema ; or it is toxæmic in its nature, as in alcoholism and gout, and thus constantly threatens the first condition of sufficient compensation. Attacks of cardiac difficulty with excitement, præcordial distress, dyspnœa, and pain are relatively common. Failure sets in early, is far more difficult to check, and readily returns—to terminate fatally. In other instances the patient perishes either of the primary disease or of one of its complications, such as cerebral hæmorrhage or softening.

Diagnosis.—As a rule the diagnosis of cardiac enlargement is easily accomplished by physical examination with the exercise of ordinary care. Two conditions, however, are a source of some difficulty. First, dislocation of the heart, and therewith of the præcordial dulness and apex-beat, might be confounded with enlargement. This is particularly likely to happen in fibrosis of the left lung, where the heart is displaced towards the left, the anterior border of the left lung is retracted, the pericardium widely exposed, and the impulse more extensive and apparently more forcible. Secondly, enlargement of the heart is sometimes inappreciable in pulmonary emphysema, and its existence may then have to be assumed from general knowledge instead of directly observed.

The presence or absence of a cardiac murmur serves to distinguish valvular and non-valvular enlargements of the heart from each other, respectively, in the stage of compensation. But in the stage of failure valvular murmurs may disappear ; and, *vice versâ*, systolic murmurs may make their appearance in non-valvular enlargement as a result of mitral or tricuspid stretching. Then the diagnosis mainly turns on the other features of the case, particularly the state of the pulse and lungs, the volume and specific gravity of the urine, and the patient's history.

Prognosis.—This varies widely, with the cause of the enlargement, as described in the section on this subject (p. 382). On the whole, non-valvular enlargement is a less favourable condition than valvular, in respect of both health and the prospect of life.

Treatment.—The main indication for the treatment of enlargement of the heart is to deal with the primary disease or disorder which lies at the root of it. When other than valvular disease, the treatment of which has been already fully discussed (p. 408), this is often directly amenable to temporary control—for example, gout, physical strain, alcoholism, chronic bronchitis, and in some degree even nephritis, arterial sclerosis, and emphysema. A new physiological balance is artificially maintained by means of rest, nourishing diet, and cardiac tonics on the one hand, and of purgatives, spare diet, and vascular depressants on the other hand, as circumstances demand. Whilst the necessity for increased action and hypertrophy of the heart is thus moderated, care is taken not to interfere with nutrition and exercise so far as to impair the myocardium. The treatment of failure of the heart in enlargement without valvular lesion is not essentially different from that which is indicated for failure in valvular disease.

RARER MYOCARDIAL LESIONS

RUPTURE OF THE HEART

Rupture of the walls of the heart with few exceptions is fatal immediately, sudden agonising præcordial or anginal pain and collapse being the striking phenomena, consequent on physical or mental excitement. If the rupture be incomplete at first, or septal in seat, life—and the same symptoms—may continue for a considerable length of time. Treatment is palliative only.

WOUNDS

Wounds of the heart as a rule are immediately fatal by syncope or collapse, but life may be prolonged for a variable time, and recovery is by no means unknown.

NEW GROWTHS

New growths cannot be diagnosed with certainty. Their existence might be suspected, but not proved, by the development

of cardiac murmurs and failure of the heart in association with malignant disease in other situations.

PARASITES

Parasites, such as echinococcus, whether in the walls of the heart, breaking into the pericardial sac, or carried into one of the chambers by the blood, are from their extreme rarity of little clinical interest.

CARDIAC STRAIN

The sound and healthy heart may be strained acutely during violent muscular effort—especially after a full meal, or slowly by laborious occupations. Far more often the valves or the myocardium will yield to mechanical stress of either kind when they are already unhealthy, as the result of mal-nutrition, toxæmia, or senility, or when they are actually diseased.

Anatomical characters.—Cardiac strain may involve either the valves, the parietes, or both. The arterial or the auriculo-ventricular valves may be variously damaged in acute strain; and they become thickened, stretched, bent, or otherwise deformed and rendered incompetent in persistent high tension. During violent muscular effort all the chambers of the heart are over-distended; and dilatation may remain permanently, accompanied by compensatory hypertrophy. The same condition is slowly developed by laborious occupations of a physical kind; and *post mortem* the myocardium usually proves to be degenerated, the coronaries thickened and tortuous, and the arterial system sclerosed, whilst the ordinary pathological characters of cardiac failure are present.

Clinical characters and course; prognosis, and treatment.—*Acute valvular strain* during effort is attended with a sudden sense of præcordial pain and anxiety. Death may occur instantly; or the patient lives, having developed an endocardial murmur, or some change in a murmur previously recognised, and probably drifts steadily into cardiac failure of a very unpromising and intractable character. Treatment must be directed, at first, to relieve the acute distress and embarrassment of the heart; later, to establish and maintain compensation.

Chronic valvular strain corresponds clinically, and in respect of treatment, with either arterial sclerosis (p. 444) or chronic valvular disease (p. 398).

Acute parietal strain of the heart from violent exertion is characterised by phenomena which are in an extreme degree the familiar appearance and distress of a person at the end of a race, particularly if he be out of training or have run upon a full meal. There is great breathlessness, a feeling of fulness and constriction over the sternum, pains in and beyond the pectoral regions, violent palpitation, and fulness and throbbing of the vessels of the neck. When the muscular effort is ended, the person may throw himself backwards and forwards to obtain relief; in extreme cases he falls, or his head drops back or hangs on his chest, the mouth is open, and gasping attempts are made to breathe more freely. The face is anxious, and livid or leaden in colour; the eyes are full, and the pupils large; the pulse fails. Physical examination discovers the signs of cardiac dilatation, including a tricuspid systolic murmur, and possibly others.

Such are the phenomena of acute over-distension and strain of the chambers of the heart, that is, acute dilatation by over-filling, caused by the inrush of a fuller and more rapid charge of venous blood from the active muscles than can be passed through the lungs and thence through the left cavities, which are themselves further embarrassed by the increased peripheral resistance that accompanies severe muscular effort. When the over-distension has reached a certain degree, however, the heart is relieved automatically in the direction of least resistance, that is, backwards into the right auricle and veins through the tricuspid orifice by "safety-valve action" of the tricuspid, unless the heart have been previously diseased, in which case death may occur immediately. When the effort is ended there is speedy recovery from the over-distension, but there remains in some instances permanent dilatation from strain of the muscular fibres.

Acute over-distension and dilatation of the heart, if it do not immediately yield to ready measures, such as fresh air, relief from tight or oppressive clothing, and the application of cold to the face, demands *treatment* with venesection, followed by cardiac stimulants, including brandy, ether, or strychnine hypodermically. Perfect rest must be ensured for several days; and the condition thereafter treated like one of chronic strain. Young subjects will have to resume exercise with great caution, and to beware of violent efforts for the rest of their life. The occurrence of acute strain in persons over

forty requires definite restriction for the future of muscular exercise, which, however, must not be entirely abandoned.

Chronic parietal strain is described under Enlargement of the Heart, as well as in connection with Arterial Sclerosis, with which it is essentially associated.

DISEASES OF THE PERICARDIUM

ACUTE PERICARDITIS

Etiology.—The efficient causes of acute inflammation of the pericardium are micro-organisms and toxins of specific febrile diseases, particularly rheumatism, scarlet fever, and septicæmia. It is also intimately associated with acute and chronic Bright's disease, and with chorea. The proximity of the pericardium to the endocardium and myocardium, and to the pleura, lungs, œsophagus, mediastinum, abdomen and chest walls, determines its invasion in many instances.

The morbid anatomy is described at p. 330.

Symptoms.—The clinical characters of acute pericarditis are both general and special. The former include fever and the symptoms proper to the so-called primary disease—rheumatism, nephritis and the like; and they therefore vary widely in different instances, including the pulse and temperature. The special symptoms of acute inflammation of the pericardium are at first those of cardiac excitement with increased action. Thereafter præcordial distress, pain, anxiety, and dyspnœa become prominent, and possibly vomiting and delirium, constituting the second stage of pericarditis; the inflammatory process, including effusion, is now at its height. Finally the phenomena of cardiac failure may set in, sometimes from associated myocarditis; but far more commonly all the symptoms decline, whether spontaneously or as the result of treatment, absorption having taken place, and the process ends possibly in adhesion—the third stage. Happily, in a number of instances the special symptoms of pericarditis are so mild as to pass unrecognised.

Physical signs.—In the first stage of the disease the præcordial impulse is extensive, powerful, heaving or jogging, and frequent; tenderness may be elicited; friction fremitus is rarely felt. The præcordial dulness is moderately increased in area. The most

characteristic sign is pericardial friction, increasing in loudness and distinctness; therewith there are heard accentuated cardiac sounds, and usually one or more endocardial murmurs. When effusion supervenes, the patient lies on his back, inclined towards the right side. The impulse becomes weaker and much more localised, or may be imperceptible in recumbency; and the apex-beat is weakened and displaced towards the left and upwards, *i.e.* in the direction of the axilla, whilst the respiratory movements of these parts are diminished—an important association. If the effusion be large the interspaces may become obliterated, the whole præcordia bulge, and the integuments be œdematous. The transverse præcordial dulness is increased in both directions—in large effusions beyond the apex-beat to the left; and the vertical dulness rises markedly, as high as the second left cartilage or even the clavicle, whilst an area of impaired resonance, varying with posture, may be developed over the lower lobe of the left lung posteriorly. The pericardial friction now becomes much less extensive, but does not entirely disappear, continuing to be heard at the base; the endocardial sounds and murmurs become less loud than before. The pulse may be paradoxical. Absorption is attended with reappearance and increasing strength and area of the visible and palpable impulse, which becomes exceedingly complex in its characters, return of the apex-beat, and increased extent and loudness, followed by weakness and disappearance, of friction; whilst the area of dulness contracts, often, however, remaining permanently high along the left border of the sternum.

Clinical course.—Acute pericarditis may pass through the different stages described; or effusion may not occur; or, on the contrary, it may persist indefinitely. Patches of roughening or thickening of the serous membrane are sometimes left, characterised by local friction sounds, which are developed or increased by pressure, particular postures, and forced expiration. The duration is indefinite. The disease terminates by recovery in the great majority of rheumatic cases, but very rarely in renal or septic cases, and then death may occur suddenly.

Diagnosis.—This is established positively by discovery of the characteristic physical signs, particularly friction. Pericarditis is simulated in its different stages by endocarditis, cardiac enlargement, and pleural effusion; and its presence is often concealed by association with these. In other instances it is obscured by the prominence of vomiting and delirium, which suggest cerebral disease. The diagnosis must always be completed by determining

the nature of the inflammation, whether rheumatic, renal, septic, etc.

Prognosis.—This turns mainly on the cause of the pericarditis: a rheumatic case will almost certainly recover, a renal or infective case not. Other unfavourable features are largeness of effusion, association with extensive pleurisy or pneumonia, high fever, cardiac failure, dysphagia, and severe chorea.

Treatment.—The etiological indications for treatment must be fully respected in pericarditis. In most instances they are fulfilled by prescribing salicylates, whilst septicæmia and Bright's disease demand correct attention to their causes respectively. The pathological indications are those proper to inflammation disposed to spontaneous resolution. Hyperæmia is controlled with light, warm applications, or with the icebag, or by means of six to twelve leeches over the sternum if pain or anxiety be great. Absorption is promoted with iodides and salines internally, and occasionally by local counter-irritation with iodine or cantharides. Very rarely is it necessary to remove the products by paracentesis, or by incision if they be purulent. The pericardium is best opened in the fifth left interspace close to the sternal border. As far as they are clinically indicated, the principal objects of treatment in the first stage are to quiet the heart by bodily and emotional rest. The attendant rheumatism of the joints usually compels the former, relief of pain and anxiety fulfil the latter object. During the second and third stages the same means must be employed to support and strengthen the heart as in the treatment of endocarditis, including proper fluid food, alcohol, ether, ammonia, digitalis, and strychnine. In all three stages morphine often proves invaluable, particularly if pain be urgent, and is best given hypodermically, combined with strychnine, *e.g.*—

R. Injectionis morphinæ hypodermicæ, ℥iij.
Liquoris strychninæ hydrochloridi, ℥iij.

—but it must be used with great care. If the heart fail acutely, venesection to eight or ten fluid ounces and the use of oxygen may avert death. As the case progresses favourably and during convalescence the treatment is modified as in endocarditis.

CHRONIC PERICARDITIS

Chronic pericarditis is met with clinically in three principal forms: (1) *adherent pericardium*; (2) *white patches* or “milk spots”

on the front of the heart; and (3) *tuberculous pericarditis* with effusion.

I. ADHERENT PERICARDIUM

The expression *adherent pericardium* is applied to a variety of pathological conditions, including loose, thick œdematous attachments of the two layers to each other, and adhesions of different degrees between the pericardium and one or more of the structures superficial to it, including four layers of pleura, the lung, and the parietes. It is commonly a result of acute rheumatic pericarditis, and associated with chronic valvular disease. When the pericardial cavity is obliterated and the different layers of pleural, pulmonary, mediastinal, and parietal structures are welded together, the so-called "signs of adherent pericardium" are found. The condition interferes with both the filling and the emptying of the cardiac chambers; disturbs the flow, distribution, and pressure of the blood within them; and leads to a degree of general dilatation and hypertrophy of the heart. The symptoms and many of the physical signs then found in connection with pericardial adhesion are characteristic simply of some cardiac disability and occasional cardiac difficulty and distress, associated with enlargement of the heart; but these are rarely diagnostic of it, being common to it with chronic endocardial and myocardial lesions. Certain signs or combinations of signs, however, have been regarded as evidence of adherent pericardium. They include permanently increased vertical dulness, systolic relapse, and diastolic rebound within the region of the apex, weakening of the true apex-beat, loss of its mobility in change of posture and in inspiration, recession of the epigastrium in inspiration, sudden diastolic collapse of the jugular veins, immobility of the anterior border of the left lung in inspiration, systolic retraction of the lower ribs at the side or back of the left chest, and unusual loudness of endocardial murmurs over the back of the chest; also the paradoxical pulse. However, these signs are so variable and so often associated with those of valvular disease and enlargement, as to be untrustworthy diagnostically; and it is but rarely that the existence of this morbid condition can be successfully determined.

The **prognosis** and **treatment** are based on the same general considerations and principles as those of chronic valvular disease.

2. WHITE PATCHES OF THE PERICARDIUM

These opacities (p. 331) possess no clinical importance. They are of clinical interest only in as far as they appear to be the source of various friction sounds met with occasionally in healthy individuals, which may be difficult to diagnose from murmurs of endocardial origin. The diagnostic signs are referred to on p. 351.

3. TUBERCULOUS PERICARDITIS

The present account of tuberculous pericarditis relates to the disease in its chronic form, the pathological anatomy of which is described on p. 335. Acute tuberculous pericarditis is commonly but a part of acute general tuberculosis (Vol. I. p. 225). Occasionally it is a local complication of chronic pulmonary tuberculosis, or the result of extension from tuberculous bronchial glands.

The **clinical characters** of chronic tuberculosis as it affects the pericardium include the constitutional symptoms of this disease; the physical signs of pericardial effusion; and, as a rule, the phenomena of similar effusion in one or more of the other serous cavities, namely, the pleura and the peritoneum. **Diagnosis** may be difficult unless this association be attentively regarded. Cardiac symptoms proper are rarely present.

The **prognosis** is essentially unfavourable. The value of local **treatment**, such as painting the præcordia with preparations of iodine to promote absorption, is doubtful. Paracentesis pericardii has been proposed, but is seldom practised.

HYDROPERICARDIUM

Hydropericardium is described under the head of Dropsy, of which it is one form. Its relation to mechanical congestion of the coronary veins has been already referred to on p. 323.

HÆMOPERICARDIUM

Intra-pericardial hæmorrhage is a result of rupture of the heart (p. 334), of wounds, and of the bursting of myocardial abscess or of aneurysm of the heart or first part of the aortic arch. Acute hæmorrhagic pericarditis has also been referred to (p. 330). Hæmopericardium is characterised by the phenomena of rapid failure of

the heart (p. 362) passing into syncope; and with rare exceptions it speedily ends in death.

PYO-PNEUMO-PERICARDITIS

The pathological characters of associated inflammatory fluid and gas in the pericardial sac are referred to at p. 331. Clinically it has been recognised by the presence of a tympanitic percussion note, shifting with change of posture, over the præcordia, and of various splashing sounds heard in the same situation (p. 352). As a rule it ends fatally.

FUNCTIONAL DISORDERS

The various symptoms referable to the cardio-vascular system which have been described in the foregoing pages have been regarded as being directly connected with definite structural abnormalities which underlie them. It is to be observed, however, that most of these phenomena may occur without any discoverable morbid alteration in the heart or vessels, and indeed they are often determined by conditions apart from the circulatory system—for example, by an unduly distended stomach, by physical exertion, or by perverted nerve-control. Such causes acting *ab extra* upon the circulatory apparatus will disturb even a perfectly healthy heart; and their effects are likely to be greater should any circumstance render that organ more susceptible, as is the case in a constitutionally “nervous” or “excitable” temperament. This also may be the case in definite cardiac disease; and the symptom-complex which any instance of structural heart disease may present includes those manifestations immediately dependent upon the structural lesion, with others, variable in degree and of uncertain occurrence, more properly to be referred to extrinsic circumstances. Since these latter may occur independently of the former group, the term “functional disorders” may without impropriety and with some convenience be employed to designate them.

The principal causes of these disorders of the action and sensibility of the heart as distinguished from structural cardiac diseases, belong to three of the classes described in the section on general etiology. The first class includes anæmia and toxæmia

—for example, chlorosis and poisoning by tobacco and uric acid. Secondly, physical agents often cause cardiac disturbance and distress without actual lesion. Muscular exertion—especially when sudden and severe—and mechanical pressure on the heart, particularly that which attends flatulence, have this effect in persons of stout habit or otherwise out of condition. The third class of causes is the most important and familiar. It comprises nervous influences, whether central, such as emotional disturbance, or reflex—of peripheral or visceral origin, particularly in persons of neurotic temperament. In many instances more than one class of causes are at work together. Thus gouty subjects may suffer from præcordial pain after exertion; and palpitation in middle-aged women is a common result of sedentary habits, domestic cares, and the approach of the menopause combined. In some apparently sound and healthy individuals the heart responds to a demand for increased action during muscular exercise, not by acting vigorously and deliberately, and thus gradually developing its capacity or becoming hypertrophied, but by falling into a state of violent excitement, palpitation, irregularity, and distress attended with dyspnoea. “Irritable heart” is the name given to this form of cardiac disorder. It has been carefully studied in army recruits, a proportion of whom break down in this way during training, and have to be discharged as unfit for the life of a soldier.

Properly speaking, functional disorders of the heart possess no associated pathological anatomy. This statement has to be qualified, however, in three respects. First, the heart may become hypertrophied after prolonged excitement and over-action; and dilatation may result from disturbance of the muscoli papillares and mitral apparatus, particularly in association with anæmia and toxæmia, which impoverish the myocardium. Secondly, some of the causes of simple disorder become, if their action be prolonged and persistent, causes of cardiac disease—for example, alcohol and gout. Thirdly, functional disorders constantly occur in the subjects of structural lesions, such as valvular disease, and indeed are responsible for much of the suffering that attends these.

Clinical characters.—The phenomena of functional disorders of the heart are exceedingly various. Some groups or types are characterised by objective disturbances of the force, rate, and rhythm of the cardiac action, which may or may not be felt by the patient. These are usually paroxysmal, occurring in attacks which are suddenly developed, and of variable but relatively brief

duration. Such are palpitation, and cardiac excitement of milder degrees; or periods of irregularity or intermittency, fluttering, or "stopping" of the heart, with failure of the pulse, after meals, in confined places, during the night, or as an effect of tea, coffee, or tobacco. In other instances tachycardia or, more rarely, bradycardia occurs. Faintness or faints are relatively common forms—for instance, in nervous anæmic youths and stout middle-aged women.

Equally common with the first group, and often associated with them, are præcordial pains in the epigastric, left sub-mammary, and sternal regions, which may develop into pseudo-angina (p. 370); sensations of oppression in the chest, suffocation, tightness, and allied forms of distress; feelings of impending death, faintness, giddiness, or semi-consciousness; and globus—a choking, full, "lumpy" sensation in the thyroid region.

Stout nervous women often complain of so-called "spasms" in the præcordial, epigastric, and left hypochondriac regions. These consist of a sense of distension or oppression, pain, fluttering, irregularity or intermittency, and faintness; they are accompanied by sighing, great anxiety, coldness of the extremities, and cold sweats; and are relieved by loud eructations of gas from the stomach. In hysterical women emotional disturbance will sometimes bring on faintness or faints, cardiac irregularity, pain, globus, sighing, and other neurotic phenomena, or purely nervous angina ending in tears.

Physical signs.—These are mainly the visible and palpable phenomena of increased, diminished, or irregular cardiac action; and the pulse corresponds. Percussion is difficult to practise, and reveals little that is abnormal; but in some instances there is increase of the transverse præcordial dulness, significant of temporary dilatation. Auscultation confirms the existence of disturbance in the sounds; and in connection with cardiac excitement systolic murmurs may be developed temporarily at one or other of the openings, as well as weak friction sounds. The disturbances of the pulse have been already described.

Course.—Functional disorders of the heart, whilst acute in their manifestations, and ordinarily of short duration, are usually recurrent in their course, and the disposition to them persists for months or more in anæmic, nervous, gouty or other subjects. Of themselves they are never fatal, but they may be associated with serious or even grave disease, and tachycardia, bradycardia, and syncope may sometimes end in death without discoverable lesion *post mortem*.

Diagnosis.—Functional disorders have to be distinguished from structural diseases of the heart. This usually can be accomplished by attentive consideration of the sex, age, history, and general appearance and disposition of the patient; the associated nervous disorders of other parts; the causes of the attacks; the characters of the attacks, particularly their severity without evidence of actual cardiac failure or of pulmonary or venous involvement; the prominence of subjective symptoms; the absence of physical signs of valvular lesions (although bruits may occur temporarily)—pp. 348 and 349; and the effects of nervine remedies and carminatives. At the same time the possibility of underlying parietal or valvular disease must never be forgotten.

Having determined that the affection is functional only, we complete the diagnosis by discovering its cause.

Prognosis.—This is favourable in respect of life, but the prospect of recurrence and persistence is considerable, varying greatly according to the cause at work and the patient's disposition and liability to neurotic and diathetic disorders. The occasional development of structural changes must also be carefully entertained in framing a prognosis.

Treatment.—The most urgent indications for treatment are usually symptomatic. Pain, faintness, palpitation, and cardiac irregularity must be relieved at once (pp. 393 and 413). Temporary benefit can usually be afforded; but recurrence will not be prevented unless the etiological indications be ascertained, by attending to the cause at work, as well as to the patient's disposition and nervous disabilities, and treatment be directed to them. How this object is to be effected in each instance does not call for more than a brief reference here. The measures employed are as numerous and as different as the causes themselves. Thus, gouty disorder of the heart calls for spare diet, regular exercise, hepatic stimulants, alkalis and iodides. On the other hand, purely nervous disturbance of the circulation may require prolonged mental and bodily rest, generous nutrition, and bromides with nervine tonics. Tea, coffee, alcohol, and tobacco must be forbidden.

CONGENITAL AFFECTIONS

Clinically regarded, abnormalities are amongst the rarest of cardiac affections. The chief interest of them is diagnostic and prognostic, treatment being seldom possible.

Symptoms.—The symptoms and physical signs vary widely with the nature and degree of the different defects and deformities described on p. 335.

When fully developed, congenital affections of the heart in children are characterised by very striking phenomena, constituting *morbis cæruleus* or “blue disease,” so called from the cyanosis which is the prominent feature. Thus, in the most important form of all, pulmonic obstruction and defect of the inter-ventricular septum, the integuments of the face, hands, and feet, the visible mucosæ and the tongue are of a blue, venous, or claret colour; and the fingers and toes are clubbed, cold, and livid, with incurved nails and liability to local necrosis. On exertion or excitement, which the patient dreads, he suffers from fits of increased cyanosis, palpitation, præcordial distress, dyspnœa, cough, hæmoptysis, and even convulsions; and he is therefore quiet, sedentary, dull and dejected, but liable to fits of temper. Exposure to cold is unpleasant, and aggravates both the local and the general symptoms; and there may be difficulty in keeping the body warm. The alimentary organs are very readily disordered; even the act of being fed, however carefully, is sufficient in some cyanotic infants to bring on convulsions, sweating, and coldness. Bodily growth and intellectual development are delayed. The blood in cyanosis proves to be concentrated, the specific gravity rising to 1070, the red corpuscles to 9,000,000 per c. mm., the hæmoglobin value to 110 or even 160 per cent, and the white blood-cells to 16,000 per c. mm.

On the other hand, cases are not uncommon in which, with characteristic physical signs of cardiac malformation, the patient has reached adult age, is well grown and developed, presents a barely perceptible tint of lividity and no clubbing of the finger-ends, and complains of no circulatory or respiratory distress, unless under exceptional conditions. Indeed, very slight malformations, such as patent foramen ovale and anomalies of the valves, may be unattended with any appreciable symptoms or signs, and remain undiscovered. Between these two extremes many varieties are met with. The patient occasionally is the subject of malformations of other organs.

Physical Signs.—These differ widely in different forms of congenital abnormalities. By far the most common is a loud, rough, washing or tearing systolic murmur, audible over most of the præcordia, and of maximum intensity either in the pulmonic area and upwards, or about the fourth left costal cartilage and downwards. But in many instances no murmur exists, nor indeed any

other abnormal physical sign or disturbance of cardiac action sufficient to establish the diagnosis. A second striking sign is basic systolic thrill. A third important feature is weakness of the second sound in the pulmonic area as compared with its characters in acquired mitral disease. The præcordia may be prominent; the area of dulness enlarged, particularly to the right as well as upwards; the impulse widely diffused, with strong apex-beat in the left nipple-line or beyond it. Ectopia cordis has its special physical signs. Other physical signs still more rarely met with include diastolic murmur and thrill in relation to the pulmonic orifice, also systolic murmur and presystolic thrill and murmur in the tricuspid area. The relations of the physical signs and combinations of signs to the different abnormalities, respectively, will be noticed presently in connection with diagnosis.

The frequency of the pulse, naturally great in infants, is much increased, along with the depth of cyanosis, during paroxysms of distress and dyspnoea. In some older subjects of cardiac abnormalities, the pulse has been found abnormally infrequent.

Course.—In cases of extreme malformation, that is, if the development of the heart be entirely incomplete, life cannot be maintained for more than a few hours or days. Many cyanotic children die before the end of the second year; others with slight abnormalities of the orifices and septa may live more than ten years. It is altogether unusual to meet with congenital heart disease at twenty; and yet some of these subjects reach middle or even advanced age, and have less distress and disability as they grow older, particularly if the circumstances of life are easy and otherwise favourable. Failure of the respiratory functions, pulmonary congestion and catarrh, and failure of the heart account for many deaths; but the most important fact in this connection is the remarkable liability of those subjects who may reach adolescence to fall victims to tuberculosis, whether general or local—particularly of the lungs.

Diagnosis.—Persistent cyanosis in an infant, child, or adolescent at once suggests malformation or other congenital affection of the heart or great vessels. The suggestion becomes a certainty when the principal symptoms and physical signs just described are discovered to be present in various associations. But it must not be forgotten that cyanosis, murmurs, and the physical signs of cardiac enlargement may all be absent in these cases. Two other points have to be considered: (1) the differential diagnosis of the individual forms of these affections; (2) the exclusion of certain morbid conditions which might be confounded with them.

(1) A positive diagnosis of the different congenital affections from each other is usually not easy, the most that can be said in individual instances being that certain physical signs afford a strong presumption of the existence of a particular abnormality; and indeed we have seen that combinations of abnormalities are the rule. Systolic thrill at the base, systolic murmur in the pulmonic area conducted to the left and upwards, and the evidences of enlargement of the right ventricle, along with some degree of cyanosis, are significant of pulmonary stenosis, probably with incomplete closure of the septa. This being the most common of all congenital abnormalities, and its clinical characters being fairly distinctive, an attempt to determine its presence or its absence should be the first step in differential diagnosis. Systolic murmur loudest in the neighbourhood of the fourth left cartilage, that is, between the pulmonic and the mitral areas, points to defect of the inter-ventricular septum, usually sequential to obstruction at the base or at one of the auriculo-ventricular orifices. A weak diastolic murmur in the pulmonic area conducted downwards into the tricuspid area suggests the diagnosis of pulmonic stenosis with defect and consequent incompetence of the valves. An aortic diastolic murmur, conducted downwards towards the mitral area, suggests a similar affection of the aortic orifice and cusps; and this diagnosis is supported by delay in the arterial pulse. A diastolic murmur or a double murmur in the pulmonic area, not conducted downwards, and the evidences of enlargement of the right ventricle, along with cyanosis, might signify patency of the ductus arteriosus. The absence of murmur and thrill, along with cyanosis and other symptoms of congenital affection, variously developed and associated, points to patent foramen ovale, or transposition of the great vessels, or other malformation little calculated to produce fluid waves within the heart. A very loud and harsh systolic murmur in the aortic area (with or without diastolic murmur), conducted both upwards and downwards over the præcordia, and audible along the spine, associated with systolic thrill and a moderate degree of dulness in the aortic area, pulsation in the vessels of the neck and head, and no cyanosis, points strongly to congenital coarctation of the aorta, provided that aortic aneurysm and a history of all the ordinary causes of aortic-valve disease can be excluded; and, if abnormal pulsation be discovered in connection with the intercostal or other parietal arteries, and relative weakness of the pulse in the lower limbs, the diagnosis is established.

(2) Certain morbid conditions which might be confounded with

congenital affections of the heart have to be carefully excluded in every instance of difficulty in diagnosis. Endocarditis and chronic valvular disease, although very rare during the first years of life, are not unknown in association with rheumatism, articular or otherwise. A loud systolic murmur in the pulmonic area in an anæmic child might falsely suggest stenosis of the pulmonary artery. A loud venous hum and irregular pulse are readily mistaken for the signs of cardiac malformation in a nervous irritable child with delicate stomach and bowels. Some young children faint, change colour, and even become convulsed from purely nervous causes. On the contrary, congenital affections may be mistaken for acquired disease if there happen to be a history of acute rheumatism in the case, which is quite possible. To avoid error, the previous personal history of the patient must be fully ascertained in every instance, and in children evidence of the occurrence of rheumatism in some form or phase particularly searched for.

Prognosis.—In individual cases of congenital affection of the heart it is usually difficult to estimate correctly the prospect of life, of health, and of ability to undertake work or even to be educated. The differential diagnosis is first to be made, if possible; and then the prognosis is to be framed on a careful consideration of the degree of disturbance and inefficiency of the heart produced by the particular abnormality believed to exist, of its course, as described above, and of the circumstances under which the patient will live.

Treatment.—The causes of cardiac malformation, being still unknown, are not within our control, and the pathological conditions are beyond repair. Therefore neither preventive nor remedial treatment is possible. The therapeutical indications are clinical only: to maintain the elementary functions of the body at the highest practicable level, and to prevent or relieve as required the attacks of cardiac distress, dyspnœa, cyanosis, and nervous disturbance to which these patients are liable. The diet must therefore be highly digestible as well as nutritious. The bowels are to be moved daily, and occasionally evacuated freely by means of saline purgatives. Cyanotic children require to be warmly clad and otherwise guarded against exposure to cold; and their liability to tuberculosis must not be forgotten. Physical rest is usually ensured automatically, but mental quiet has to be definitely ordered because of the natural irritability of the subjects of congenital anomalies of the heart. Cardiac failure is treated on the same principles as in acquired disease.

DISEASES OF THE BLOOD-VESSELS

DISEASES OF THE ARTERIES

The subject of the disorders and diseases of the arteries possesses much more than immediate or local importance. The arteries are physiologically related to the heart, to the peripheral resistance, and to the blood; and their pathological relations with each of these are equally intimate, and extend far beyond the limits of the vessels themselves. Thus it has been shown in a preceding section how the size, tension, and condition of the vessel-wall, as elements of the pulse, are variously affected by every disturbance and disease of the heart; how arterial degeneration produces hypertrophy of the left ventricle; and how often morbid changes in the arteries are associated with those forms of enlargement of the heart which are independent of valvular lesions, as for example in chronic nephritis. The influence of pathological conditions of the blood as causes of arterial lesions has been described in Vol. II. It follows from these considerations that the position which diseases of the arteries occupy in Pathology and Practical Medicine must not be estimated solely by the lesions peculiar to the vessels themselves. Still, it will be presently learned that these also are of great importance.

GENERAL ETIOLOGY

(1) Some of the principal causes of arterial disease belong to the group of *micro-organisms and toxins* associated with the infective processes, particularly influenza, typhoid fever, syphilis, tuberculosis, and septicæmia, as well as with suppuration in the immediate neighbourhood of the vessels. (2) Other diseases of the arteries are effects of *anæmia* and *toxæmia* in different forms and of different kinds, notably chlorosis, local anæmia produced by disease of the *vasa vasorum*, gout, diabetes mellitus, alcoholism, and plumbism. (3) Of equal or even greater frequency and importance in the production of pathological changes in the walls of the arteries are certain *physical* influences. Thus stress is seen at work in the form of violent muscular efforts and laborious occupations, in the increased peripheral resistance of Bright's

disease, and in chronic gout and other disorders of metabolism connected with free living and deficient elimination. Traumata are of great variety, including wounds and other injuries of arteries from without, and damage of them from within by embolism, by thrombosis, and by the sudden violent alternate stretching and relaxation to which their walls are subjected in aortic incompetence. (4) Extremes of *heat and cold* readily give rise to arterial lesions. (5) The preceding causes frequently act in different *combinations*. Thus gout both impoverishes and poisons the vascular walls, and strains them by raising the peripheral resistance; and the arterial sclerosis of senility is to be attributed to anæmia, to increased peripheral resistance produced by withering and obsolescence of many of the capillaries, to failure of nervous energy, and to the natural exhaustion of vitality.

GENERAL MORBID ANATOMY AND PATHOLOGY

ARTERIAL DEGENERATIONS include hyaline and fatty degenerations, calcification, and amyloid disease. The three first of these are local changes, often associated with atheroma and arterial sclerosis, and will be described in connection with these. Amyloid degeneration is a more diffuse process, which does not call for notice here.

ARTERIAL THROMBOSIS.—Coagulation of the living blood is an element of several pathological processes involving the arteries. It is familiar as the result of wounds of the vessels, including surgical ligature, where it constitutes one of the various provisions for repair and permanent closure. Chronic lesions of the arterial walls, for example, aneurysm, atheroma, obliterative arteritis, arterial sclerosis, and syphilis, readily develop local clotting; and this may lead to serious interference with the local circulation and the nutrition of the tissues around—for example, in the brain and heart (p. 334)—and possibly to rupture. Embolism of arteries also is followed by thrombosis, the clot extending centripetally to the nearest branch, at which it may cease to grow, or which it may invade. If the embolus have been non-infective, the thrombus becomes organised: fibro-blasts grow from the intima, penetrate the clot, and become developed into connective tissue, which is vascularised from the *vasa vasorum*. The result is complete or partial obliteration of the affected part of the vessel, behind which dilatation or rupture may occur. If the embolus have been infective, the thrombus breaks down, in association with purulent

infiltration of the walls and possibly acute aneurysm. Besides its local effects in interfering with nutrition, arterial thrombosis sometimes gives rise to embolism by detachment of parts of the clot; and thus thrombosis and embolism are mutual causes of each other.

ARTERIAL EMBOLISM.—The sudden occlusion of an artery by an embolus, a particle of clot or necrotic tissue carried from another part of the circulatory system, is characterised by a series of important pathological changes, including infarction, thrombosis, and their effects. Impaction of the foreign body in an end-artery is immediately followed by complete or incomplete necrosis of the elements of the tissues supplied by the vessel—*pale infarct*, embolic softening. When the process is accompanied, as it is in other situations, by capillary engorgement and abundant diapedesis of the red blood-cells into the same parts, the result is the development of a *hæmorrhagic infarct*, a deep-red swollen area of a pyramidal or conical shape, with its narrow extremity at the seat of interruption of the circulation. At the same time a thrombus forms behind the embolus. If the latter be non-infective, the hæmorrhagic infarct gradually becomes decolorised, the thrombus is organised, and the affected area as a whole shrinks and undergoes fibroid change. On the other hand, if the embolus contain pathogenetic organisms—derived, for example, from the heart in malignant endocarditis, or from the veins in septicæmia—the infarct, arterial walls, and thrombus rapidly undergo purulent infiltration, soften and break down, and the result is either a hæmorrhage, a septic abscess, or an acute infective (embolic) aneurysm, which may afterwards rupture.

ARTERITIS.—Inflammation of arteries may be either acute or chronic. When the necrotic and degenerative factors are in the ascendant the process is acute. The vascular walls become swollen, thickened, and softened by necrosis and infiltration with pus-cells and micro-organisms. As a result, *acute infective aneurysm* is developed, or the walls give way and hæmorrhage occurs—familiar terminations of arteritis in tuberculous lesions of the lungs and in gastric and intestinal ulcers. In other instances they actually suppurate. These are common results in septic embolism, and are also met with where an artery is in relation with an inflammatory or tuberculous focus. On the other hand, when the cause is non-infective, the reparative factor is comparatively powerful, and the arteritis is plastic and usually chronic. The several coats, especially the intima, are the seats of active cellular proliferation, and the vessel as a whole becomes thickened, cord-like, and lengthened. This process is accompanied by formation

and organisation of a thrombus, and contraction or even obliteration of the lumen is the result. The latter pathological change is fully developed in *end-arteritis obliterans*—where the intima becomes enormously thickened by infiltration with young cells and their development into connective tissue, whilst the media is changed but little, and the adventitia considerably (Fig. 30). When it involves the circulation extensively or universally, particularly the vessels of the kidneys and brain, chronic proliferative arteritis constitutes *arterial*

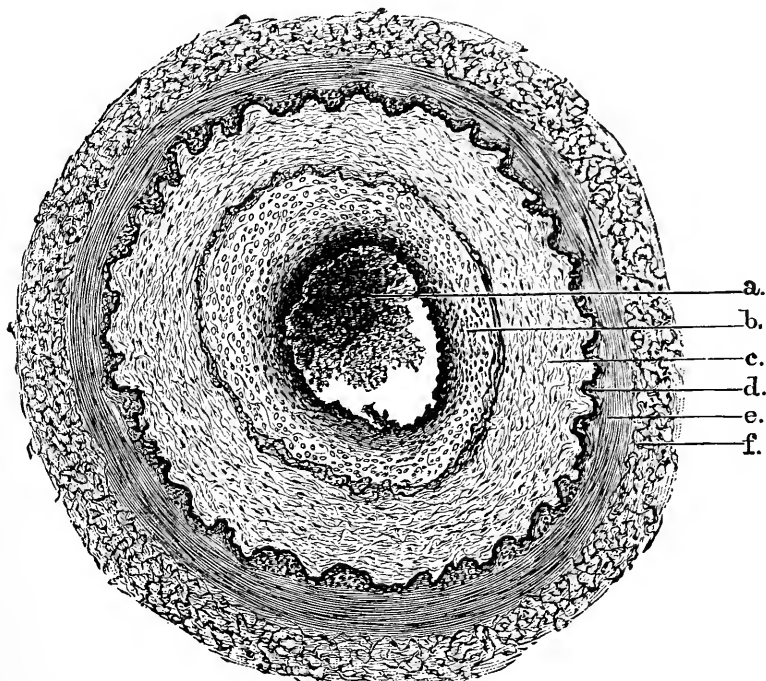


FIG. 30.—Arteritis obliterans, syphilis of brain. *a*, blood clot; *b*, inner layer of thickened intima; *c*, outer layer of the same; *d*, inner elastic lamina; *e*, muscularis; *f*, thickened adventitia. $\times 50$. (Hamilton.)

sclerosis or *arterio-capillary fibrosis*. The intima presents local thickenings or elevations which have a yellowish-white smooth surface unless they be thrombosed, and are mainly formed by an abundant growth of connective tissue, which may become vascularised from the *vasa vasorum* and grow by fresh thrombosis. *Syphilitic arteritis* of the smaller vessels mainly involves the intima. This is greatly thickened by the presence of germinal cells, which develop into fibrous tissue (Fig. 31). The adventitia is similarly infiltrated; the media is comparatively unaffected. In the aorta and other large vessels syphilitic arteritis takes the form of circum-

scribed grayish-white nodules in the intima or adventitia, which proceed to sclerosis, whilst the media is also puckered from

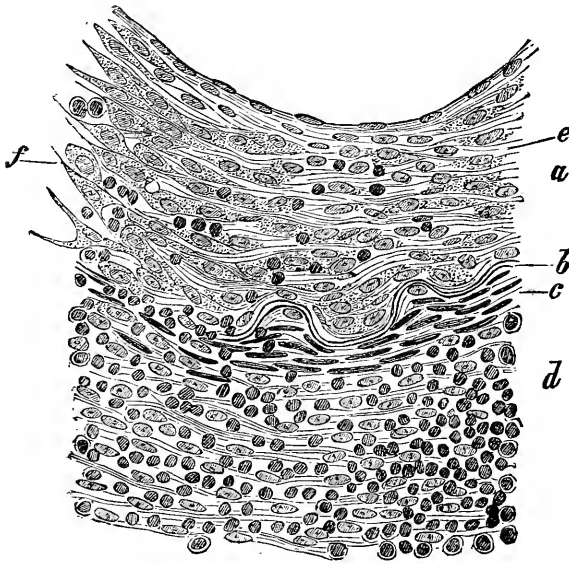


FIG. 31.—Syphilitic arteritis. *a*, Intima greatly thickened; *b*, fenestrated membrane, broken towards the left; *c*, muscularis; *d*, adventitia; *e*, fibro-cellular tissue; *f*, young cellular tissue. $\times 150$. (Ziegler.)

cicatricial contraction of nodules around the *vasa vasorum*. Still another form of arterial inflammation is known as *periarteritis nodosa*. This process produces small nodules or local swellings on the vessel, visible to the naked eye, and involving all three tunics. Thrombosis may be associated, and the disease may also spread into the surrounding tissues.

ATHEROMA.—Atheroma is recognised as elevated, yellowish patches or spots in the intima of the aorta (as

well as in the endocardium—p. 326) and the vessels arising from it, particularly at their origins; also in the smaller arteries, and in the pulmonic arterial system when the mitral valve is diseased. These patches are formed by proliferation of the fibrous and elastic tissues of the intima and media, associated with fatty and hyaline degeneration of the elements—including the muscular fibres, and only occasionally fibrosis of the adventitia also. At first atheromatous spots have a somewhat translucent or gelatinous appearance, but afterwards they become opaque, cartilaginous-looking, and possibly calcified (Fig. 32). In other instances they soften, contain a creamy, puriform material, consisting of degenerated elements, oil-globules, cholesterin, etc. (*atheromatous abscess*); and finally they may break into the lumen, forming an *atheromatous ulcer*, covered or not with thrombi, whilst the *débris* is washed into the circulating blood.

HYPERTROPHY of the walls of arteries is characterised by increased thickness and length of the vessels, with consequent tortuosity. Both the muscular and connective tissues are involved. This change is typically met with in old age, and in some cases of chronic Bright's disease.

ARTERITIS

Of the causes of arteritis the most important are the acute and chronic infective diseases, particularly septicæmia, syphilis, and tuberculosis, poisons such as lead, anæmia, embolism, thrombosis of different kinds, wounds (including ligature), mechanical stress, senility, and valvular lesions. These and other influences of a less

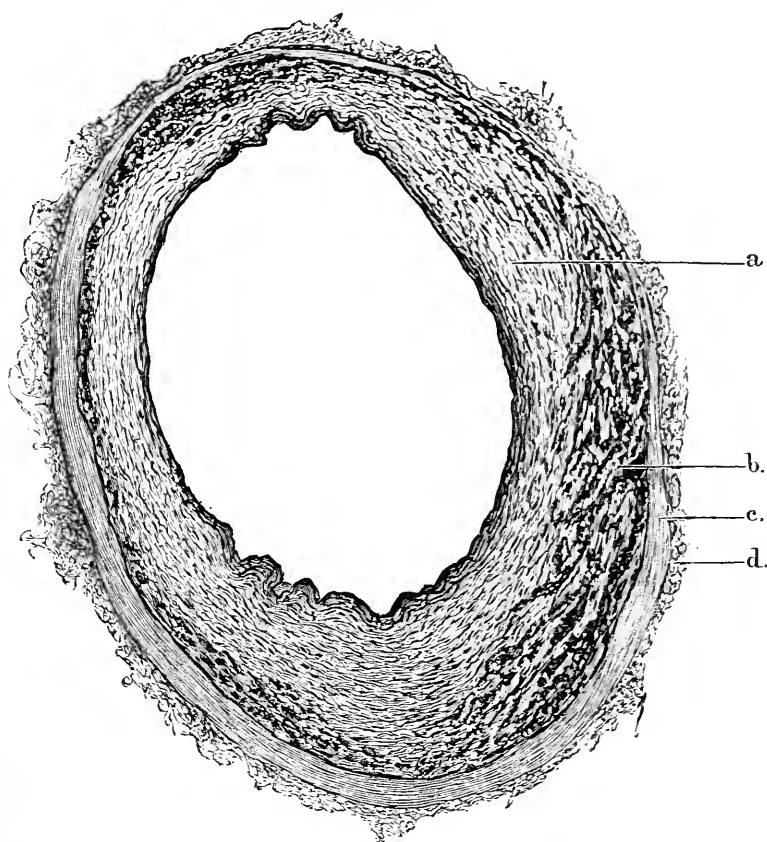


FIG. 32.—Atheroma. *a*, Unilaterally thickened intima; *b*, atheromatous part of the same; *c*, muscularis, attenuated opposite the atheromatous part; *d*, adventitia, comparatively unaltered. $\times 40$. (Hamilton.)

definite character set up inflammatory changes, partly degenerative, partly regenerative, in the intima, media, and adventitia, as well as thrombosis. The relative degrees of involvement of the different coats and the lumen, and of the severity of the different pathogenetic causes of arteritis, lead to the great variety of changes in the arterial walls, which have been described above.

ACUTE ARTERITIS

LOCAL ACUTE ARTERITIS starts either from without, as when an artery is involved in an area of suppuration, or from within, around a thrombus or an infective embolus. In either case it may lead to aneurysm or hæmorrhage, or both. Its clinical characters and course, and the related prognosis and treatment, are described in connection with Arterial Embolism.

GENERAL ACUTE ARTERITIS is a very rare disease. It occurs as a complication or sequela of fevers, particularly influenza and typhoid, and is called "general," because any part or more than one part of the arterial system may be involved—particularly the vessels of the lower limbs. All the three coats are involved; the intima bulges into the lumen; and thrombosis occurs. Clinically, the diagnostic evidences of the disease are the discovery of this thrombosis; its effects in interrupting the circulation—coldness, sensory disturbances, pulselessness, œdema, and gangrene of the limb; its association with a specific fever; and therewith pain and tenderness along the course of the affected vessels.

CHRONIC ARTERITIS

Chronic arteritis occurs in the form of obliterative arteritis and arterial sclerosis.

OBLITERATIVE ARTERITIS.—This disease originates in local thrombosis, in non-infective embolism, in syphilis, in tuberculosis, and possibly in other chronic affections. Its morbid anatomy has been described above. The **symptoms** which characterise it are different according to the cause at work and the organ involved. Thus, obliterative arteritis of the brain is one of the chief pathological changes underlying the complex phenomena of cerebral syphilis, and obliterative arteritis of the pulmonary system is associated clinically with tuberculosis of the lungs and hæmoptysis; when it affects the coronary vessels it leads to myocardial degeneration and possibly rupture of the heart; in the limbs it causes gangrene. The **prognosis** and **treatment** vary accordingly.

ARTERIAL SCLEROSIS.—Under this name there are grouped a variety of pathological changes in the cardio-vascular system characterised by the common features of chronic arteritis and enlargement of the heart.

Etiology.—This disease possesses great importance etiologi-

cally. It is associated with disordered metabolism; with the actions of certain poisons, such as lead, alcohol, and uric acid; with advanced age; with prolonged strain of the circulation, and with chronic renal and cardiac lesions. At other times it is the outcome of acute and other specific processes, particularly influenza, syphilis, and typhoid and scarlet fevers. Possibly nervous stress accounts for some cases of arterial sclerosis. In many instances it is due to a combination of these causes, especially in soldiers; and in still other cases its etiology is entirely obscure.

Morbid anatomy.—The pathological characters of arterial sclerosis are described above (p. 441). The vessels most commonly affected are those of the brain, heart, and kidneys, and the smaller arteries of the limbs. At the same time it must be observed that some authorities include under this head not only the generalised disease produced by the above causes, but also the localised or discrete form of arterial degeneration commonly called atheroma (p. 442). The heart is dilated and hypertrophied, the enlargement advancing from the left ventricle backwards through the left auricle to the right side. Failure has usually set in before death. In addition to the morbid changes directly associated with the causes just enumerated, pulmonary emphysema and cerebral hæmorrhage or softening are often found *post-mortem*.

Symptoms.—The clinical characters of arterial sclerosis are as various as are the causes of the vascular degeneration in different instances. When it arises from mechanical stress the disease is commonly described as *chronic cardiac strain*, and the symptoms and signs are those of dilatation of the heart with compensatory hypertrophy, ending, after a variable time—usually years—in ordinary cardiac failure. The radial pulse is large, thickened, tortuous, possibly locomotive, and of variable tension; the rhythm of the pulse-wave also varies according to the stage and other features of the case, and its amplitude is diminished. Capillary pulsation may be present. In other instances of this affection the prominent clinical characters are those of chronic gout, of chronic alcoholism, of cerebral thrombosis—whether syphilitic or not—of senility, of tabes, of valvular disease, of aneurysm, or of emphysema with dilatation of the right heart, respectively.

Whatever its origin may be, arterial sclerosis is characterised by symptoms and signs referable to the different viscera involved. Præcordial oppression, pain of an anginal type, faintness, and dyspnœa are significant of affection of the coronary arteries, and consequent impoverishment or degeneration of the myocardium;

whilst systolic murmur in the aortic and mitral areas points to degenerative lesions of the valves, and the signs of enlargement are also present. Cerebral symptoms are very common, corresponding with anæmia from vascular degeneration. They include impairment of memory and vertigo, passing paresis, insomnia, depression, headache, and confused ideas of time and place; on which there may supervene at any time the symptoms of thrombosis and cerebral softening. A large and important sub-group of general arterial sclerosis is constituted by the arterio-capillary fibrosis of chronic interstitial nephritis. Pulmonary and bronchial affections, with enlargement of the right side of the heart, are often associated with the preceding. The state of the blood and of the body as a whole is impoverished; debility increases; and dropsy gradually develops, particularly when the kidneys are involved.

Arterial sclerosis commonly ends in cardiac failure; other terminations are sudden death from fatty degeneration of the heart, uræmia, cerebral hæmorrhage or softening, and rapid failure in acute disease or after operations.

Diagnosis.—The difficulties attending the diagnosis of arterial sclerosis from valvular disease are discussed at p. 421, in connection with the subject of enlargement of the heart.

Prognosis.—The complexity of this subject is obvious, the prospect of life and health varying widely with the cause of arterial degeneration and the organs chiefly affected. Speaking broadly, the prognosis is extremely unfavourable, life insecure, and the possibility of sudden death to be remembered.

Treatment.—Arterial sclerosis is to be prevented mainly by temperance in eating and drinking, by the avoidance of syphilis, by the rational enjoyment of exercise, by moderation in work, both muscular and nervous—in a word, by observance of hygiene in general. When the disease is established, its causes must still be recognised and dealt with if possible—particularly syphilis, gout, and physical and mental stress—by the employment of proper measures respectively. The pathological indications plainly include the avoidance of cardio-vascular excitement on the one hand, and of depression on the other—that is, the observance of a quiet, even, temperate mode of life, including small digestible meals with sparing stimulants; regular but gentle exercise of muscles and brain; and at the same time improvement of the blood and nutrition by well-selected food, attention to digestion, and regular elimination by bowels, skin, and kidneys. Iron, arsenic, strychnine, and small doses of digitalis and the iodides, as occasional drugs, may be of

considerable use, as well as baths and massage in some cases. Cardiac failure must be treated on recognised principles when it occurs.

EMBOLISM

As an occasional incident in the course of certain diseases of the heart and blood-vessels, a portion of the products of inflammation or degeneration is detached by the force of the blood-stream or by bodily movement and swept into an artery, where it becomes impacted. The immediate result is arterial occlusion at the point of arrest, and disturbance of the nutrition both of the walls of the vessel, of the branches of the same, and of the tissues which they supply, ending in infarction, thrombosis, and their effects. The particle thus conveyed and arrested is called an *embolus*; and the pathological process, as a whole, embolism.

Etiology.—In arterial embolism the sources of the embolus are the veins, the heart, or the arteries themselves. Most commonly when derived from a vein, emboli are portions of thrombi associated with plastic or infective phlebitis, which, if not arrested in the heart, as may happen when they are large, are carried into the pulmonary system (p. 76, Pulmonary Embolism). Emboli coming from the heart consist either of particles of thrombi or necrosed tissue from the valves, the products of simple or ulcerative endocarditis or of degeneration; or of thrombi from the auricles or ventricles, the results of cardiac failure. When brought from the right chambers of the heart, they produce infarction of the lungs; those which originate in the left chambers of the heart are carried into the arteries of the viscera—particularly the spleen, brain, kidneys, and intestines—or of the limbs; sometimes the abdominal aorta itself is blocked by large masses. Emboli of arterial origin are derived from the sacs of aneurysms or are portions of thrombi formed in connection with arteritis.

In rare instances embolism is produced by irruption of foreign bodies or materials into the circulation. Such are parasites, portions of new growths, and inflammatory products—for example, the purulent contents of abscess of the heart. In this connection it may also be mentioned that air is sometimes admitted into veins through wounds in the neck, and fat as a result of fractures, with embolism of the heart and pulmonary vessels as the result.

Anatomical characters.—The morbid anatomy of arterial embolism is described above.

Symptoms.—The symptoms of arterial embolism vary widely, both with the nature of the primary disease and embolus, and with the seat of the lesion. Its actual occurrence is frequently manifested by sudden disturbance of the functions of the organ involved, possibly amounting to complete arrest. Thus paralysis is a familiar symptom of cerebral embolism, with or without apoplexy; sudden death may result from coronary embolism; impaction of a clot in the main artery of one of the limbs is attended with severe pain, disappearance of the pulse, coldness, and paresis; pulmonary embolism gives rise to pain, dyspnœa, hæmoptysis, and pleuritic friction; embolism of the *arteria centralis retinæ* disturbs vision, and its phenomena can be observed with the ophthalmoscope. In connection with some of the viscera special symptoms are developed—for example, albuminuria and hæmaturia in renal embolism, violent abdominal pain and hæmorrhage from the bowel in mesenteric embolism, and distress in the left hypochondrium when the spleen is affected. Subsequent symptoms point to the damage caused by infarction, such as permanent hemiplegia, gangrene of a limb, rupture of an embolic aneurysm within the abdomen, or hopeless loss of vision. In many cases embolism is multiple, particularly when the primary disease is ulcerative endocarditis.

Prognosis.—In non-infective embolism the gravity of the prognosis is in direct proportion to the size of the vessel occluded. The prospect of local recovery in arterial embolism is far more favourable in parts provided with free arterial anastomosis and ready collateral circulation than in those which possess end-arteries; but complete recovery of the function of the damaged area is rare. Infective embolism is but one element of a disease which almost necessarily proves fatal within a short time.

Treatment.—Impaction of an artery as a pathological condition is not amenable to remedial treatment. Every effort should therefore be made to prevent it in diseases which may give rise to it. Neither the establishment of collateral circulation nor the disappearance of the embolus can be hastened therapeutically. The primary disease demands great attention, especially when it is of an infective nature.

ATHEROMA

Atheroma is a complex pathological process, involving the arterial walls, of which chronic arteritis and degenerations are the principal factors. It is one of the results of chronic circulatory

strain, senility, high tension, gout, plumbism, of local tuberculosis, and of syphilis, individually or collectively. Sometimes it can be traced back to acute arteritis due to fevers. Further, it begets itself by involving the *vasa vasorum* of the larger arteries.

The morbid anatomy of atheroma has been described above.

The grave effects of this disease, in the form of stenosis, thrombosis, or actual obliteration of arteries, with consequent softening, fatty degeneration, and necrosis of the tissues which they supply, are illustrated in the preceding sections on diseases of the heart and vessels. These have been referred to under degeneration of the myocardium and degenerative disease of the valves, arising in connection with atheroma of the coronary arteries, the mouths and trunks of which it often involves.*

Atheroma of the *aorta* is recognised by physical signs, by the characters of the radial pulse, and by certain prominent symptoms. An area of abnormal percussion dulness is found over the manubrium, and this may extend downwards until it fuses with the normal præcordial dulness. Pulsation may be visible at the sternal end of the second right interspace, and visible and palpable in the suprasternal notch. The first sound in the aortic area is commonly replaced by a coarse systolic murmur; the second aortic sound is loud and ringing, and possibly hollow. Therewith there are found the ordinary signs of hypertrophy of the left ventricle. The radial pulse is large, of variable tension; the vessel wall is thickened, possibly calcified, tortuous, visible, and locomotive; the pulse-wave is regular, irregular, or intermittent, of ordinary frequency, sudden, ample, and slow. The subject of atheroma is usually a person of middle or advanced life, with a history of one or more of the diseases which have been mentioned as the causes of it. He may complain of præcordial oppression, palpitation, dyspnœa on exertion or during the night, and sometimes of anginal pain. Symptoms of defective cerebral circulation and of myocardial weakness are occasionally associated; and the disease terminates as a rule by the occurrence of grave disease of the brain or heart. Yet many old persons live for years with the signs just described, and in good health but for passing attacks of præcordial distress and dyspnœa when the circulation is taxed.

The **prognosis** of atheroma is similar to that of arterial sclerosis, to which it is essentially related. The same remark applies to the preventive, remedial, and palliative **treatment** of this disease.

ANEURYSM OF THE THORACIC AORTA

Etiology.—The efficient cause of aortic aneurysm is high arterial pressure, whether absolute or relative. The determining circumstances that give effect to this intra-vascular stress are chiefly sudden exertion and laborious occupations. But an intrinsic factor is rarely absent, namely, previous damage and loss of natural resistance of a part of the aortic wall, consequent on chronic arteritis—particularly syphilitic, or previous injury the result of stress or trauma; and so considerable may such damage be that even the ordinary blood-pressure is sufficient to originate aneurysm.

Anatomical characters.—Aneurysm is a local dilatation of an artery in its course, produced either by stretching of all its coats or by rupture of one or more of them.

The aorta is the favourite seat of aneurysm—in any part, from the sinuses of Valsalva to its bifurcation, the thoracic aorta and the arch in particular being most often involved. But every artery, whatever its size, is liable to this disease, and the pulmonary artery and its divisions suffer similarly.

Aneurysms have been variously classified by their form, the constituents of their walls, their relations with other vessels, their course, their situation, etc. Thus, when a considerable length of an artery is dilated more or less uniformly, the resulting enlargement is *fusiform*; when the dilatation is lateral and in communication with the lumen of the artery by a definite opening, it is called a *sacculated* aneurysm. If one or more of the three vascular tunics still enter into the structure of the wall of the swelling, by virtue of being stretched, the aneurysm is said to be *true*; if they have given way, and the surrounding tissues or newly formed connective tissues constitute its boundaries, it is said to be a *false* aneurysm. Whatever be the constituents of its walls, an aneurysm may be definitely bounded—*circumscribed*; or, if it be false, its contents may be extravasated in irregular spaces in the tissues, constituting *diffuse* aneurysm. Occasionally an aneurysm that has started from rupture of the intima and part of the media becomes diffuse in the walls of the artery itself by tracking along it between the layers of the parietal tissues—*dissecting* aneurysm; such an aneurysm may finally break outwards by rupture, or break inwards—back into the true lumen. In other instances an aneurysm forms a communication with a neighbouring vein—*aneurysmal varix* and *varicose aneurysm*.

Fusiform aneurysms are best studied in connection with the arch of the aorta. They are of every variety of size (length and girth), some, for example, involving the whole of the arch; they are usually irregular, presenting smaller dilatations upon the general enlargement, and they may be associated with local sacculations.

The *constituent parts* of an aneurysm are fully developed in the sacculated form, which is that most commonly met with in the thoracic aorta. It presents a body or sac, walls, a neck, a mouth or opening into the artery, and contents. The *sac* may be of almost any size; it is either regular and single, or irregular with local depressions or secondary sacs. The *walls* are lined with a smooth endothelial coat, but are often extensively atheromatous. The *neck* is usually short, the aneurysm being sessile on the vessel wall. The *mouth* is usually regular and smooth, and varies greatly in size in different instances. The *contents* of an aneurysm are either fluid arterial blood, red clot, or laminated "fibrin," or thrombi in vascular connection with part of the lining membrane, and themselves more or less organised.

Associated pathological changes.—Along with aneurysm there occur other important pathological changes. The rest of the aorta may be extensively diseased. Aortic valvular disease is a frequent accompaniment; and then the left ventricle is enlarged, not otherwise. Dislocation of the heart, disease of the coronary arteries with consequent myocardial degeneration, pericarditis, and intra-pericardial hæmorrhage from rupture are other morbid conditions occasionally met with. The innominate, carotid, and subclavian arteries are variously diseased, kinked, dislocated, or embolised. The great veins within the chest and in the neck are obstructed, distended, varicose, and possibly thrombosed. The thoracic nerves are often involved in the aneurysm, particularly the left vagus and recurrent laryngeal, the intercostals, and the branches of the cardiac and pulmonary plexuses. The lungs undergo three important changes: (1) direct compression; (2) collapse, due to pressure on the trachea or bronchi; and (3), rarely, destruction consequent on septic broncho-pneumonia originating in inhalation of foreign materials through the paralysed larynx. Perforation of the bronchi or trachea and hæmorrhage are often found to have been the immediate cause of death. Pleurisy is not uncommon. Hæmothorax is sometimes found. The œsophagus may be dislocated, compressed, or perforated. The bones and muscles of the chest, including the spine, are sometimes involved in the sac, which may have penetrated the parietes and become diffused

under the integuments, or actually ruptured externally. Distant organs like the brain and spleen may be the seat of embolism and its results.

Symptoms. — In a case of well-marked aortic aneurysm the patient is commonly a man of early middle age, who has followed a laborious occupation—very often that of a soldier—and has suffered from syphilis. He complains of pain in the chest, dyspnœa, beating of the heart, and increasing weakness; and he presents an anxious, weary, dusky appearance, with fulness of the face and neck, visible and audible dyspnœa, a broken or hoarse voice, and clanging cough. Thoracic *pain* is of two kinds. The first is a fixed pain, referred to some part of the chest-walls, front or back, increased by exertion and during the night, possibly associated with tenderness. The second is intermittent neuralgia along the course of the intercostal and brachial nerves, occasionally anginal, with tingling and other disturbances of sensation. The distribution of this referred pain varies with the situation of the aneurysm. If the arch be the portion involved, the third and fourth cervical, or the first, second, third, or fourth dorsal areas, are painful and tender; if the descending aorta be aneurysmal, the fifth, sixth, and seventh dorsal segments are the seat of disordered sensibility. But it is important to observe that the pain of aortic aneurysm may be strictly limited to the terminations of the intercostal nerves. *Dyspnœa* is also of two forms in aortic aneurysm: namely, first, dyspnœa on exertion, after cough, or in uncomfortable postures; and second, paroxysms of orthopnœa, with anxiety and lividity, often unfortunately confounded with bronchial asthma. In both forms the dyspnœa is usually stridulous. The *voice* is often husky or cracked; occasionally there is aphonia. Besides occasional moderate *cough* there may occur seizures of a sudden, violent, spasmodic, and clanging character, broken by long, loud, and stridulous inspirations, and leading to alarming agitation of the chest and congestion of the veins and face. Expectoration may follow, the *sputa* being either white and frothy, or in thick, tenacious masses, purulent or blood-stained. Small *hæmoptyses* occur in many instances from moderate leakage into the pulmonary alveoli; but if a bronchus or the trachea be invaded, a sudden and fatal outburst will presently occur, or this may happen instantly without previous warning. Fatal hæmorrhage occurs in other instances into the œsophagus, pleural or pericardial cavities, or elsewhere. True *dysphagia* from œsophageal obstruction is a classical but by no means a frequent symptom. Interference with the first

stage of swallowing—the passage of food over the larynx—is more common; it may give rise to paroxysmal dyspnoea and cough during meals, inhalation, and septic broncho-pneumonia. *Spinal symptoms*, such as painful paraplegia, from slow compression or more serious involvement of the cord, are relatively rare. The *temperature* is not disturbed, unless by complications such as secondary broncho-pneumonia. Even septicæmia may then occur. The general nutrition varies with the severity of the disease, that is, of the pain, insomnia, dyspnoea, difficulty of swallowing, and distress. Exhaustion and wasting sometimes advance rapidly, and death may occur from asthenia instead of from one of the many dangers constantly threatening life.

Physical signs.—The following description relates to fully developed aneurysm of the thoracic aorta. The patient's chest being fully exposed in a good light, which falls obliquely across the surface, inspection reveals a *pulsating area or tumour*, independent of the præcordial impulse and of pulsation in the carotids and subclavians. This is usually in front, rarely behind, and most commonly in relation with the upper half of the sternum, or the three upper cartilages on either side of it; its extent, outline, surface, etc., vary greatly; occasionally it is absent. By palpation, which is assisted by full expiration in the stooping posture, we discover that aneurysmal pulsation consists of a true impulse or heave in cardiac systole, and a diastolic shock, which are often much more powerful than the same signs over the heart. Sometimes there is also thrill, usually systolic, rarely a rippling sensation; if the aneurysm be large, the pulsation is expansile. Pulsation is also palpable over the trachea or larynx. In some instances *tracheal tugging* is discovered. If the observer press the cricoid cartilage lightly and steadily upwards with his finger and thumb, he becomes aware of a gentle tug on the trachea and larynx downwards, synchronous with each cardiac systole. The neighbouring parietal veins are prominent and tortuous, and can usually be traced from similarly distended veins in the neck and in one or both arms; but this is far from being a constant sign. Corresponding œdema is not uncommon. The pulsating area is dull on percussion, and the limits of dulness are characteristically separate from those of præcordial (cardiac) dulness, but the two are sometimes continuous. The auscultatory signs over the seat of aneurysm are, first, systolic sound—of different characters—and diastolic shock-sound, the second sound being intense, ringing, intoned or booming, and thudding or accompanied with a distinct sensation of agitation

conveyed to the ear and head ; and, secondly, in many cases systolic and diastolic murmurs of great variety. The systolic murmur is believed to be either valvular or aneurysmal in origin ; the diastolic is practically always valvular. Very similar auscultatory signs are found over the base of the heart proper.

Certain other physical signs must next be determined, to ascertain the seat and characters of the apex-beat and the sounds there ; also the existence or not of compression of the lungs and bronchi, of pulmonary collapse, and of pleural effusion. The pulse should be tested in all the great arteries, and thereafter at both wrists. The characteristic radial pulse is unsymmetrical in size, force, and time (delayed) ; the sphygmogram corresponds. The abductor of the left (rarely the right) vocal cord is found to be paralysed. Inequality of the pupils is sometimes met with, from interference with the cilio-spinal nerves in the upper aperture of the chest. Search should next be made for superficial tenderness by Head's method. The œsophageal sound must never be used to investigate the cause of the dysphagia.

Course.—Aortic aneurysm is a chronic disease, of indefinite duration, uncertain in its progress, full of possible complications according to the direction of its growth, and greatly influenced by the conditions of the patient's life. If the growth and increase of pressure on neighbouring structures be backwards, upon the bronchi and œsophagus, both distress and danger are far greater, although the signs may be indefinite, than when the tumour becomes more prominent and more alarming to the inexperienced observer. Many cases improve temporarily under treatment, but even these, like the others, ordinarily die suddenly of hæmorrhage in the course of a few years.

Diagnosis.—This includes determination of: (1) the existence, (2) the precise seat, and (3) the form, of aortic aneurysm.

(1) The *existence* of aneurysm is diagnosed by consideration of all the etiological and clinical facts already described ; but it must be understood that in some instances many of them are wanting, or, indeed, all of them, whether symptoms or signs, and that the diagnosis may then be extremely difficult or even impossible. The diseases which simulate it are chiefly aneurysms of other vessels ; tumours of the chest-walls ; new growths involving the pulmonary or the mediastinal tissues or the œsophagus ; tuberculosis, and other wasting and fibrotic affections of the lungs, with retraction of the anterior borders, displacement and visible pulsation of the aorta, and palpable diastolic shock ; syphilitic stricture of a main

bronchus, with possibly secondary phthisis; congenital coarctation of the aorta (p. 436); and associated aortic incompetence and chronic laryngitis, both syphilitic. Much greater difficulty attends the diagnosis of small aneurysms of the aorta close above the sinuses of Valsalva, and of deeply seated aneurysms, for instance, of the descending aorta, the symptoms—dyspnoea and wasting in particular—being often referred to asthma, and the radiating pains to intercostal neuralgia, whilst the physical signs are practically absent or so ill-marked as to be unreliable. The X-rays may then be of great service; and they should be employed in all doubtful cases.

(2) The diagnosis of the *precise seat* of aortic aneurysm is mainly a matter of acquaintance with the topographical anatomy of the chest, but certain of the clinical phenomena may be mentioned as specially useful in regional diagnosis. Pulsating tumour to the right of the sternum in the neighbourhood of the third cartilage is significant of aneurysm of the ascending part of the arch—possibly intra-pericardial; and this diagnosis would be confirmed by an aortic diastolic murmur with cardiac enlargement, anginal pain on the right side, and signs of obstruction of the right internal mammary vein (p. 358) and possibly of the superior cava. Disturbance of the right pupil, referable to involvement of the second dorsal sympathetic ganglion, points to an aneurysm near the upper extremity of the ascending portion. Signs of pressure on the right bronchus—including diminished vocal fremitus and weak respiratory murmur, without impairment of resonance, over a part or the whole of the corresponding lung—should be carefully searched for as being evidence of involvement of the junction of the ascending and transverse parts of the arch. These signs may be accompanied by distension of the veins of the neck and upper part of the chest, drained by the right vena anonyma, and a small weak delayed right radial pulse. Pulsation in the supra-sternal notch, distension of the veins that fall into the left vena anonyma and œdema of the areas which they drain, dulness over the manubrium and inner end of the left clavicle, urgent stridulous dyspnoea with lividity on exertion, tracheal tugging, and the signs of a degree of collapse of both lungs—these phenomena point to involvement of the summit of the arch. One of the most useful guides to the exact locality of an aortic aneurysm is paralysis of the left recurrent laryngeal nerve and vocal cord as observed with the laryngoscope. This phenomenon and signs of compression of the left bronchus are evidences of affection of the third part of the arch. Physical signs

of forward dislocation of the heart, dysphagia, pains referred to the lower parts of the chest and upper parts of the abdomen, paraplegia, and dulness with pulsation or a pulsating tumour in the left scapular region, are severally and collectively diagnostic of aneurysm of the descending thoracic aorta.

(3) The diagnostic features of the two principal *forms* of aortic aneurysm are as follows:—

FUSIFORM ANEURYSM

1. Signs more diffuse.
2. More thrill.
3. Systolic murmur harsh, rasping, and loud.
4. Pressure symptoms extensive, but less marked individually.
5. Pressure signs do. do.
6. Local tumour and perforation of chest-wall less likely.

SACCULATED ANEURYSM

1. Signs more localised.
2. Less thrill.
3. Systolic murmur soft or absent. Possibly *not* systolic when accurately timed.
4. Pressure symptoms few, but severely marked.
5. Pressure signs do. do.
6. Local tumour and perforation of chest-wall more likely.

Prognosis.—The general prospect of life in aortic aneurysm may be estimated by the facts described under “Course.” In individual cases the prognosis will be based on the immediate urgency of the symptoms, such as hæmoptysis and dyspnœa; on the evidence of backward pressure *versus* forward growth (p. 454); on the patient’s circumstances and temperament; on the association of other aneurysms, cardiac disease, and arterial sclerosis; and on the effect of treatment. The uncertainty of life and the probability of sudden death must never be forgotten.

Treatment.—The principal indications for the treatment of aortic aneurysm are etiological: to lower arterial pressure, to remove all the conditions of life calculated to raise it, and to deal with syphilis, which is so often the cause of the loss of resistance in the arterial walls. These indications are best fulfilled by means of rest, low diet, removal of discomfort and anxiety as far as possible, and the administration of potassium iodide in the largest doses that can be borne. Rest must be absolute in bed, the patient rising for no purpose whatever. Diet should be reduced to about 8 or 10 ounces of solids and 10 to 15 fluid-ounces of liquids per diem. The iodide is often given in 20-grain doses four or six times a day. With the use of rest and low diet in aneurysm the name of Tufnell of Dublin, who first used them systematically, is worthily associated. Success attends these combined methods in many instances, particularly of the sacculated form, pain, dyspnœa, and discomfort

all steadily diminishing and disappearing, pulsation becoming much weaker, and the patient returning to a degree of comfort and useful activity for a time. In other instances modified rest is employed, along with large doses of iodide, particularly if the patient be of an impatient temperament, and likely to fret rather than be contented under enforced confinement to bed.

Palliative treatment is usually called for, at least until the patient has been fully brought under the influence of the more radical measures. Ordinary paroxysmal dyspnœa is relieved with ether and ammonia; sudden severe attacks of it may require brief inhalations of chloroform; grave and prolonged seizures are treated with venesection, which often gives great relief and rescues the patient from imminent death. Pain that does not yield to potassium iodide, or to an ice-bag over the tumour, demands morphine hypodermically. Various expectorants lighten cough and expectoration. Small hæmoptyses may cease under treatment with strict rest and quiet, and the use of cold externally and internally. It is essential that the bowels should be gently moved daily.

Various surgical methods of treatment have been employed in aortic aneurysm, but hitherto with little success. They include the introduction of sterilised horse hair, fine wire, etc., into the sac, with the intention of provoking a deposit of fibrin upon the foreign body and consequent consolidation; electrolysis; and a combination of the two measures. Injections of a 2 per cent sterilised neutral-saline solution of gelatin (3 to 8 fluid ounces every few days) into the gluteal muscles have been followed by consolidation of the sac in some instances; but this method of treatment is not unattended with danger.

DISEASES OF THE PULMONARY ARTERY

DEGENERATIONS.—Fatty degeneration and amyloid disease have been found in the branches of the pulmonary artery.

THROMBOSIS.—As it occurs in the pulmonic circulation, thrombosis is usually caused by retardation of the blood stream, consequent either on failure of the heart, whether rapidly or slowly developed, or on local compression of the trunk or branches of the pulmonary artery by tumours, including thoracic aneurysms. Anæmia and other disorders of the blood, embolism, and local disease of the

arterial walls are also factors of coagulation here, as in the vessels of other organs.

The symptoms and signs of pulmonic thrombosis, including dyspnœa, cough, hæmoptysis and cardiac distress, are not sufficiently different from those of the diseases which cause it to be distinctive.

EMBOLISM.—Embolism of the pulmonary artery occurs in two principal forms: namely (1) embolism of the trunk or main divisions; and (2) embolism of one of the smaller branches.

(1) *Embolism of the trunk or main divisions* of the pulmonary artery can usually be traced to thrombosis of one of the larger systemic veins, as it occurs for example in typhoid fever. A large thrombus becomes detached from the femoral or iliac vein, or inferior cava, and after being swept through the right auricle and ventricle, and probably bent, folded, or coiled on itself, is arrested at or beyond the pulmonary valves. The *symptoms* are characteristic. The patient, who usually is under treatment for thrombosis secondary to one of the fevers, or is recovering from a serious pelvic operation or convalescing *post partum*, is suddenly seized with intense dyspnœa, collapse, and a sense of constriction or other form of distress across the chest. The pulse fails, until it disappears at the wrist; the countenance is pallid and anxious; a cold sweat breaks out over the body; convulsion occurs in some instances, and may be repeated. Dissolution threatens, and may immediately take place. More often the patient survives the seizure, and passes into a condition characterised by tumultuous action of the heart, in some instances with a loud systolic pulmonic murmur; by a small, irregular, frequent pulse; and by continuous and severe dyspnœa and præcordial distress, associated with loud respiratory murmur, perfect pulmonary resonance, and the absence otherwise of physical signs of disease of the lungs. These phenomena may continue for hours or for days, whereupon they either decline or pass into those of embolism of the smaller branches of the pulmonary artery, which will be presently described, including pain in different parts of the chest, hæmorrhagic sputa, dulness, and crepitant râles.

(2) *Embolism of the smaller branches* of the pulmonary artery is caused by either infective or non-infective thrombi derived from the systemic veins or from the right chambers of the heart. Infective thrombi, originating in septicæmia of the systemic or the portal veins, or in malignant endocarditis of the right side of the heart, give rise to infarction and acute suppurative arteritis in the lungs, as described above (p. 440), along with broncho-pleuro-pneumonia and pulmonary septicæmia. This affection runs a very different

course in different instances, like general septicæmia, of which it is an element; and it usually ends in death. On the other hand, aseptic thrombi, reaching the smaller branches of the pulmonary artery, produce non-infective embolism and infarction, characterised by well-recognised symptoms and signs (see Pulmonary Infarction, p. 188), one of the later complications of cardiac failure, from which the patient rarely recovers. This affection is occasionally a result of embolism of the trunk of the pulmonary artery, as already described.

ARTERITIS.—*Acute* inflammation of the pulmonary artery is most commonly either infective or tuberculous in its nature. In both cases the changes in the lumen and walls of the vessel are similar to those in the systemic arteries (p. 440). Aneurysm, rupture, and hæmoptysis are occasional results.

Chronic inflammation of the pulmonary artery and its branches is a result of persistent high tension in the lesser circulation, and is therefore a common accompaniment of emphysema and mitral disease, particularly mitral stenosis. Whilst the vessels are dilated and variously degenerated, their walls are thickened, as in chronic arteritis of the systemic circulation. More localised changes of a similar character are met with in the arterial branches supplying tuberculous or otherwise diseased areas of lung; and it is by a process of *obliterative arteritis* that the vessels in relation with tuberculous cavities usually become impervious. Should their closure be incomplete, or should a portion of the wall be weakened by the inflammatory or tuberculous processes, *rupture* may result, or *aneurysm of the pulmonary artery* be developed; and in either event hæmoptysis usually occurs.

ATHEROMA.—Atheroma of the pulmonary artery and its branches is found along with dilatation and chronic arteritis in advanced cases of mitral disease.

EROSION AND PERFORATION.—These lesions of the pulmonary artery are results of the pressure of thoracic aneurysm, of invasion by new growths, or of the spread of softening and ulcerous processes in tuberculosis of the lungs or mediastinal glands.

DILATATION.—Dilatation of the trunk and primary branches of the pulmonary artery is met with in two different forms. The first of these consists in a more or less uniform enlargement of the pulmonary vessels, with thickening of their walls, patches of atheroma, and possibly incompetence of the valves. It is associated with mitral obstruction and with emphysema, and is obviously referable to abnormal rise of pressure in the lesser circulation.

Clinically this condition is characterised by accentuation of the second pulmonic sound, sometimes followed by a soft, faint diastolic murmur in or near the third left intercostal space, midway between the left sternal border and the left vertical nipple line. Occasionally there is visible pulsation in the pulmonic area. The second form is local or irregular, and consists in sacculated or dissecting aneurysm, or in more than one. It is the result of chronic arteritis. The clinical evidences of this rare disease are principally those of thoracic aneurysm in the second and third left intercostal spaces, with marked lividity.

ANEURYSMS OF THE SMALLER BRANCHES, already referred to under Arteritis, consist of dilatations of vessels in relation to the walls of tuberculous cavities. They may be so small as to be just visible to the naked eye, but more commonly they are as large as peas, and sometimes are much larger. Occasionally many such aneurysms are found in one lung. The single clinical evidence of their existence is hæmoptysis, which often proves immediately fatal.

STENOSIS.—*Congenital* narrowing of this artery is described at p. 336. In the same connection mention may be made of other congenital abnormalities of the origin, mouth, and valves of the pulmonary artery and related parts, including patent ductus arteriosus. *Acquired* stenosis, a very rare condition, is usually referable to local arteritis, to pressure by tumours, or to fibrosis or other deforming disease of the lungs.

DISEASES OF THE VEINS

In the preceding description of diseases of the heart, reference has been incidentally made to certain morbid conditions to which they give rise in the neighbouring or otherwise closely associated veins. The most important of these affections are distension and permanent dilatation, valvular incompetence and thrombosis of the jugulars, and varicosity of the veins of the thoracic and abdominal parietes with reversal of the current within them, consequent on obstruction of the great venous trunks. Here there remain to be considered various pathological conditions of the venous walls, more particularly thrombosis and phlebitis.

THROMBOSIS

The **causes** which promote coagulation of the blood within the veins during life, and the nature of the process of thrombosis

as far as it is known, are discussed in connection with the Diseases of the Blood (Vol. II. p. 315). The first of these causes is circulatory obstruction or retardation of the blood-stream, particularly in the veins of the lower extremities and in the cranial sinuses. This can commonly be traced to weakening of the heart by exhausting diseases like carcinoma, tuberculosis, influenza, typhoid fever, and marasmus. Local thrombosis of similar origin is met with involving the subclavian vein in advanced failure of the heart. The influence of the second factor of thrombosis, alteration in the intima of the vessel, whether vein or venous sinus, is seen in connection with injuries, particularly surgical wounds (*e.g.* after extensive pelvic operations), and with parturition; and the same factor sets up thrombosis in the walls of abscesses. The third condition of intravenous coagulation is some change in the blood, or the entrance into it of foreign materials, particularly toxins or micro-organisms. That this is a common event is proved by the nature of the primary disease, *e.g.* gout, chlorosis, septicæmia, influenza, and typhoid fever, whilst tuberculosis and cancer probably act partly in the same way as causes of thrombosis. These pathogenetic agents induce coagulation within the vein by damaging either the blood-corpuscles or the endothelium of the intima, or both; and if they be of an infective nature, purulent phlebitis is the result, and occasionally septic embolism.

PHLEBITIS

Etiology.—Of the causes of phlebitis the most common is thrombosis originating in one or other of the ways enumerated above, particularly in connection with gout, carcinoma, and tuberculosis. Partly through the medium of a thrombus, partly perhaps *viâ* the *visâ vasorum*, the walls of the veins are invaded by the micro-organisms or toxins of septicæmia, influenza, syphilis, and local tuberculosis. From without, the adventitia is often injured by wounds, or it becomes involved in inflammation affecting the surrounding tissues, or in other morbid processes, such as syphilis and tuberculosis.

Morbid anatomy.—Phlebitis is of two kinds, plastic and suppurative. *Plastic* phlebitis is occasionally the result of chronic inflammation of the surrounding tissues, which spreads into the adventitia, sets up fibrous periphlebitis, and thereafter may penetrate as far as the other coats. More commonly it starts from a non-infective thrombus lying in one of the veins of the pelvis

or the lower limbs, or in one of the cranial sinuses; involves the intima and media, which are first infiltrated and then permanently thickened; and is associated with organisation of the thrombus. Stricture, or even complete obliteration, of the vein is the result, and thus the process as a whole closely resembles plastic arteritis. Resolution, however, is not uncommon. *Suppurative* phlebitis usually begins from without—by infiltration of the adventitia with micro-organisms and inflammatory cells from a neighbouring abscess. In other instances the disease originates in an infective thrombus in the lumen of the vessel, from which the walls are invaded by micro-organisms, the morbid process rapidly leading to necrosis, softening and suppuration of the affected part, and not infrequently to septic embolism of the lungs or malignant endocarditis of the right side of the heart.

Symptoms.—The phenomena of phlebitis vary widely with its cause and morbid anatomy, and with the part of the body involved. *Plastic* phlebitis is best studied as it occurs in the lower limb, sequential to pelvic disease or to influenza. Here it is characterised by local and constitutional symptoms of moderate severity, of subacute course, and usually favourable termination. These include pain, which begins in the calf of the leg; swelling, heat, and tenderness of the limb; œdema of the foot and subcutaneous parts; a cord-like condition of the saphenous or the femoral vein, with slight redness of the integuments over it, whilst the limb as a whole is unnaturally pale; and moderate fever and its many attendant phenomena. In the course of a few weeks, under proper treatment, either resolution occurs, with disappearance of all the symptoms and signs of disease, or the affected vein is left permanently stenosed or obliterated, and the limb enlarged and œdematous in spite of collateral anastomosis.

Infective phlebitis is a grave disease constituting an important factor of septicæmia. It is also met with in the portal system and liver—pyle-phlebitis; and in the cranial sinuses and the internal jugulars, which are infected in median otitis.

Treatment.—The principal indications in plastic phlebitis are to promote resolution and repair. This object is to be attained by means of complete bodily and local rest in bed, elevation of the affected limb, and the application to it of warm, moist, mildly antiseptic boric acid fomentations. At the same time the cause receives therapeutic attention by administration of ammonia, potassium iodide in moderate doses, quinine or strychnine in

different instances. The diet is usually fluid at first, but light solids are ordered as the fever declines. Stimulants have to be cautiously employed. Infective phlebitis demands the treatment proper to septicæmia.

J. MITCHELL BRUCE.

CEDEMA

SYN.: DROPSY—ANASARCA—ASCITES¹

The morbid condition to which these terms are somewhat indiscriminately applied, consists in an excess of fluid in the ultimate lymph spaces of the tissues, or in the serous sacs (peritoneum, pleura, etc.). Inasmuch as normally all these spaces are moistened by lymph which is derived from the blood in the capillary vessels, and is finally poured into the venous system by the thoracic and right lymphatic ducts and also in part reabsorbed from the spaces by the blood capillaries themselves, it would follow that an accumulation of this fluid must be due to a want of adjustment between its production and its removal.

THE NORMAL PRODUCTION AND REMOVAL OF LYMPH.—It is necessary to say a few words upon this subject preliminary to the consideration of what obtains in disease, and it will be most convenient to refer separately to each of the factors concerned.

(a) *The ultimate lymph spaces* are the lacunar interstices in the connective tissue which is distributed throughout the entire body, constituting the framework and support of the more actively living elements, epithelial, muscular, and nervous. Owing to this extensive arrangement of the connective tissue, the contained interspaces form a more or less definite communicating system which permeates every part. The interstices themselves are partially lined by flattened connective-tissue cells, which are continued on as the inner coating of the lymphatic capillaries, which take origin to some extent from these spaces. Other forms of connective-tissue corpuscles are to be found lying in the spaces, which they in part or completely fill. Whilst no direct communication appears to exist between the lymphatic vessels in their course, and these lymph spaces, the serous sacs freely open into adjacent lymphatics by means of distinct apertures or stomata.

(b) *The lymph*.—In general characters and composition the lymph resembles a very dilute blood plasma, from which it is in great part derived. Any analysis can only be approximately correct for the lymph as a whole, since it certainly varies in the different situations from which it may be obtained, and probably also in the same situation at different times. As a general average it may be reckoned to contain about 3 to 5 per cent of solids, by far the greater part being proteids (2 to 4

¹ Edema from οἰδέω=I swell. Dropsy from ὕδωρ=water, and ὤψ=aspect. Anasarca from ἀνά=through, and σάρξ=flesh. Ascites from ἀσκός=a leathern sac or bottle.

per cent), of which 0.1 per cent consists of fibrinogen, as compared to 0.4 in blood; about 1 per cent of salts, chiefly of sodium; and minute traces of fats and glucose. Since the blood, except in the spleen, nowhere comes in direct contact with the living tissue elements, the lymph forms, as it were, an intermediary, conveying from the blood to the tissues the materials required for their nutrition, and also in turn receiving the products of their metabolism which require to be got rid of. It is to these circumstances that the varying composition of the lymph is mainly to be attributed, even rendering it less constant in this respect than the blood. A few leucocytes are always to be found in lymph, the number being increased after its passage through the lymphatic glands. It is faintly alkaline in reaction, and forms a clot on standing, like the blood. Reference has been made elsewhere (p. 310) to the large amount of lymph in the body, considerably exceeding that of the blood, notwithstanding that the quantity at any one time in the terminal spaces is exceedingly small.

(c) Concerning *the blood* from which the lymph is derived, it suffices to mention its frequent variation in quantity and quality in any region, as well as the changes which occur in its rate of flow, and in the pressure that it exerts on the containing vessels.

(d) *The blood-vessels*.—The actual passage of material out of the blood-current into the lymph takes place in the capillaries, the walls of which are composed of a single layer of fusiform, and branched, flattened, endothelial cells continuous with those lining the arteries and veins. Between the adjacent cells is an intercellular substance, demonstrable by suitable reagents, somewhat thicker at places forming the “stigmata,” “which are analogous to the ‘pseudo-stomata’ found between the epithelium cells of a serous membrane. Branched cells of the surrounding areolar tissue are found connected intimately with the cells forming the capillary wall,”¹ the reticulated processes of the cells constituting in some situations a continuous covering to the capillaries and small vessels. The septum, therefore, between the blood and the cavity of the lacunar interstices will consist of the capillary layer of cells, with or without an adventitious layer formed from the supporting (retiform) connective tissue, and an incomplete layer of endothelium lining the lymph spaces. It is needful to realise that the cells are living protoplasmic structures, and experimental evidence goes to show that the permeability of the septum normally varies in different regions of the body, being much greater in the capillaries of the viscera than in those of the limbs, and is very different for different substances; and also that this same quality is liable to be affected by the presence in the blood of various substances which have hence been denominated “lymphagogues.”

Such being the factors concerned in the production of lymph, it is

¹ Prof. Schäfer in *Quain's Anatomy*, 1891, p. 370.

now necessary to consider how transudation¹ of material is effected from the blood into the lymph spaces, and by what means the lymph, when formed, is moved onwards into the lymphatic vessels to be finally passed into the main venous trunks.

Much difference of opinion has prevailed as to the precise *mechanism of lymph formation*, and which of the several factors enumerated is chiefly responsible. Whilst some (as Heidenhain) would regard the process as essentially one of secretion, in which the capillary endothelial cells play the part of a secreting gland, and the effect of variations in blood pressure as subordinate thereto; others (as Ludwig and Starling) consider that it is chiefly a matter of filtration brought about by alterations in the blood pressure, and in the osmotic pressure of the fluids concerned, though insisting at the same time upon the integrity of the septum being maintained; and to this extent only regarding the "vitality" of the tissues as being involved. A third group of observers (as Dr. Lazarus-Barlow) attribute more importance to the attraction excited by the living elements of the tissues, and see in their nutritive demand a force which determines a flow of material towards themselves from the blood. It is most probable that no one of these agencies is solely effective, but that all take a share in bringing about the result, although perhaps an actual secreting power on the part of the intervening cells is the most doubtful, and filtration is the most important. Any influence that may be exerted by the nervous system would be mainly by affecting the calibre of the blood-vessels, and consequent alteration of the quantity of blood, the velocity of its flow, and the pressure exerted by it in any particular area. The possibility of a more direct action upon the living cells of the capillary wall and endothelial lining of the spaces, with consequent modification in the quantity and quality of the lymph produced, may not be forgotten, although no proof in this direction is available.

The removal of lymph.—The means by which the lymph, when formed, is moved on *via* the lymphatic vessels into the main venous trunks are of a twofold character. "The first and chief factor in the onward flow of lymph is the pressure under which this is formed in the radicles of the lymphatics and in the tissue spaces. As the blood flows

¹ The term "transudation" is used to express the passage of a fluid through a porous substance; the passage of fluid through a membrane consequent on a difference of hydrostatic pressure on the two sides is "filtration"; the intermixture taking place between the watery part of two liquids, which are separated by a membrane, determined by differences in composition or specific gravity in the liquids, in opposition to hydrostatic pressure, is termed "osmosis"; as *dialysis* refers to the interchange of substances held in solution. When the membrane itself exerts an active (vital) influence upon the selection of the material that passes through it (from the blood) the process is known as "secretion." The word diffusion refers to the intermixture of two indifferent fluids when brought into contact (*i.e.* without any intervening septum), due to the constant movement of their constituent molecules.

through the capillaries at a given pressure, a certain proportion of its fluid constituents filters through the vessel wall, forming a transudation which is still under a certain amount of pressure, and it is this remaining pressure which causes the onward flow of the lymph. Hence the ultimate cause of the lymph flow must be looked for in the energy of the heart's contraction."¹ Accessory to this, though probably on the whole the most effective, are the movements of respiration and of the muscles generally. Consequent upon the pressure being diminished in the thorax and increased in the abdomen during inspiration, the contents of the receptaculum chyli and abdominal lymphatics are sucked onwards into that portion of the thoracic duct which lies in the chest, and this is still further aided by the aspiration exerted on the main lymph channels by the negative pressure in the intra-thoracic venous trunks during inspiration; with expiration, the pressure in the chest being increased, the lymph is forced onwards into the veins, the valves in the lymphatics and main ducts preventing a backward flow. The anatomical arrangement of the lymphatics in respect to the tendons and enveloping fasciæ of the systemic muscles is such as to be most favourable to the onward flow of the lymph when the muscles contract. Exercise, therefore, becomes an important agent in maintaining the movements of the lymph, as it is of the blood (p. 310), it being the fact that there is scarcely any lymph flow in a part that is at rest.

But the removal of lymph is not solely effected by the lymphatics, for it has been shown experimentally that the capillaries, whence the lymph is derived, are also effective in removing the same by absorption from the tissue spaces, and with comparative rapidity. Variations in the intra-capillary blood-pressure and velocity will influence the extent of this absorption, as they do the previous transudation, though of course in the reverse way. How far the septum (*i.e.* capillary wall and adjuncts) exerts any active influence is a most important point, as it was seen to be in regard to the formation of lymph. It seems certain, however, that the condition of the septum does influence the extent to which the various ingredients of lymph are reabsorbed into the blood. It is doubtful to what extent a difference exists between the lymphatics and blood-capillaries in the lymph constituents which they severally remove, but it is certain that proteids are absorbed with difficulty, and perhaps chiefly reach the blood *viâ* the lymphatic channels.

Thus, then, the lymph which normally bathes the ultimate tissue interstices and the interior of the serous sacs, whilst continuously being derived from one set of vessels—the blood-capillaries—is as continuously removed by two sets of channels—the lymphatics and the blood-capillaries—and, further, the combined capacity of the latter is

¹ See article on the production and absorption of lymph, by Prof. Starling, F.R.S., in *Text-Book of Physiology*, edited by Prof. Schäfer, F.R.S., vol. i. 1898, to which the reader is referred for detailed information on this subject.

much greater than that of the supply. Hence, in a normal condition, the channels of removal being more capacious than those of formation, no accumulation of fluid takes place, and it is not until the means of removal becomes from any cause less in carrying power than the production, whether this be excessive or not, that any accumulation can occur. The pathology of œdema, therefore, resolves itself into an investigation of the determining causes of the disturbance of this balance between production and removal.

Pathology of œdema.—From what has been already said it is clear that the causes and modes of disturbance of the several factors concerned in the normal production and removal of lymph must be first considered. But it is important to understand at the outset that a condition of œdema is seldom the result of a perversion in one factor only; the intimate association in function of the several structures involved determines sooner or later a disturbance in those which may not at first have been impaired. In this way the one morbid state intensifies the other, until it may be difficult to see where the vicious circle commenced.

The blood.—Just as the production and absorption of lymph is normally governed by the character of the blood, its specific gravity, its viscosity and its composition, and by the velocity of its flow and the pressure that it exerts in the capillary vessels, so these, when abnormal, become the chief causes that lead to œdema or dropsy.

One of the most frequent conditions with which œdema is associated is a simple anæmia or chlorosis. Swelling of the feet and ankles, especially after standing, and relieved more or less after the legs have been raised to the level of the trunk, is so frequent as to be expected; and an artificial hydræmia induced by replacing a known quantity of blood in a living animal by a corresponding amount of normal saline solution is quickly followed by an increase in the lymph produced, which, if not removed, accumulates in the lymph spaces. But even in so apparently simple a case as this it is impossible to eliminate the possibility of some alteration in the permeability of the capillary wall being brought about by the alteration in its nutrition due to the impoverished blood, and so possibly permitting a greater transudation than otherwise would have occurred. Still more is this likely to be the case when the blood, whether more watery than normal or not, contains a large proportion of crystalloids, either of those usually present, such as sodium chloride, glucose, etc., or altogether foreign, such as potassium iodide (Heidenhain's second class of lymphagogues). By some it has been sought to show that the action of these in increasing the lymph formation is due to the higher osmotic co-efficient¹ of these

¹ By osmotic pressure is signified the pressure exerted by the water which passes through the membrane as the result of the molecular movements of the substances in solution on the other side of the same, *i.e.* as the result of osmosis.

substances in solution over the substances present in normal blood-plasma, and that their mode of action is by attracting fluid from the tissues into the blood and so producing a state of hydræmic plethora in which the intra-capillary pressure is much increased, which has been demonstrated experimentally to lead to a very greatly increased lymph flow (Starling). The general anasarca that so characteristically accompanies some forms of renal disease in which the blood is profoundly altered is probably in a great measure to be explained in this way, although it is quite probable that the quality of the capillary wall is also impaired, having regard to the known tissue changes which occur in these affections. It is recognised that certain forms of toxæmia, as some of the acute infections, autogenetic poisoning from the alimentary canal, poisoning by tissue fibrinogen, as well as by certain substances taken with the food, or special articles of diet, *e.g.* mussels, all probably of the nature of peptones or albumoses (corresponding, that is, to Heidenhain's first class of lymphagogues) are liable to give rise to œdema, often of very variable distribution, but sometimes quite local, as the lip, face, or hand. In such cases it does appear that the lymph accumulation is chiefly due to some alteration in the capillary wall, whereby an excessive transudation takes place; and Heidenhain would attribute it to increased secretory power on the part of the cells. The influence of blood pressure at any rate is not so obviously effective in such cases, especially as it is often lowered in these conditions.

The condition of the blood circulation.—Of as great an importance in the production of œdema as the quality of the blood is the degree of pressure which that fluid exerts upon the capillary wall, for upon that depends very largely the amount of filtration which takes place. Obviously the most effective method of raising the capillary pressure in any area is to obstruct the venous return from the same, provided the *vis a tergo* be not diminished, and the experimental ligature of veins, if extensive, is commonly followed by increased lymph production. Similarly such morbid conditions as lead to obstruction and consequent plethora of the venous system, whether this be general, as in regurgitation at the tricuspid orifice, or affecting the pulmonary circulation only, or chiefly, as in mitral stenosis, or the local effects of venous thrombosis or pressure upon venous trunks, each and all lead to the occurrence of œdema. But although there is this close association of dropsy with venous obstruction, as seen both experimentally and in disease, it nevertheless seems to be clear that it is not the obstruction alone that brings about œdema, but that there must be at the same time some impairment of the vessel wall, *i.e.* of the septum. For although venous obstruction may raise the intra-capillary pressure, it does not apparently do so in a state of cardiac failure, when the force of the left ventricle is very much lessened, and moreover œdema has been known to follow the ligature of the main artery to a limb when the blood force in the arteries of the affected region

falls.¹ Experimentally also the venous blood pressure in a limb has been kept for some hours, twelve or more times greater than normal, with no appreciable œdema as a result. In many cases of obstructed circulation due to congenital heart affections there is a remarkable absence of dropsy, all tending to suggest that there must be some other cause at work than modifications of blood pressure, important as these may be, and that this is to be found in the character of the septum through which filtration takes place, and, further, that when this septum is structurally normal no excessive transudation occurs. The capillary pressure will also be raised when a venous plethora is induced by excessive absorption into the vessels; and such substances introduced into the blood, *e.g.* dextrose, as will cause an increased absorption from the tissues, ultimately lead to an increased effusion by the fulness of the venous system which they have at first induced.

Indirectly, increased intra-capillary pressure may be considered to favour accumulation in the lymph spaces and serous sacs by diminishing the completeness of the removal of the lymph when formed. Such a condition would be adverse to the absorption by the capillaries, at the same time that it favoured excessive transudation, until the extra-capillary pressure should have risen very considerably, and this again would the sooner tend to occur, if the venous obstruction were cardiac in origin, by interfering with the ready escape of the lymph into the venous trunks.

When from over-production with defective removal the lymph accumulates and distends the spaces, the pressure therein rises, and so tends to equalise the conditions on the two sides of the membrane, and consequently arrests further transudation; thus it is that in long-standing cases of venous obstruction the resulting œdema remains at very much the same amount for considerable periods, and may even somewhat diminish from the greatest measure which has been attained.

The tissue elements.—Premising that it is impossible to regard the share taken by the tissue elements in the production of dropsy apart from the existing characters of the blood and conditions of the circulation, just as it is impossible to regard the effect of the blood pressure quite apart from the nature of the membrane through which filtration occurs, it is nevertheless desirable for a full consideration of the pathology of this state to look at the subject from the point of view of the living cells of the tissues concerned, since it is essentially on their account that the lymph is formed, and into them that the products of their metabolism are in great part thrown.

Reference has been made to the all-important part played by the cells of the capillary wall, the resistance they offer when unimpaired to any excessive transudation, the damage they may undergo from malnutrition, degeneration, or direct toxic effects, permitting a filtration

¹ The intra-capillary pressure, however, does not necessarily correspond with that in the arteries (see p. 308).

under alterations in blood pressure. There is also some evidence to support the view that they take an active share in effecting the œdema comparable to the process of secretion.

Besides these are the living constituents of the tissues, cells of various kinds and muscle fibres; and Dr. Lazarus-Barlow, by careful comparisons of the specific gravity of the blood and of the tissues under altered conditions of circulation with and without œdema of the part, concludes that this condition depends upon "an excess of the normal process by means of which the normal nutrition of the tissues is maintained. . . . Upon this view the tissues would primarily determine the occurrence of dropsy and not the blood-vessels."¹

In the tissue response to irritation, known as inflammation, œdema is one of the phenomena; and although doubtless the condition of the circulation in the affected area is altered, and the blood pressure, at least for a time, increased, it is most probable that the capillary walls have undergone some change, and the altered vitality of the tissue elements is undoubted. The excessive nutritive demands under the altered circumstances would, according to the view just set forth, be the principal cause of the associated œdema, determining an increased secretory action on the part of the cells. The differences in the inflammatory effusions met with in peritonitis, pleurisy, and pericarditis respectively have also been held as evidence of a selective (secretory) activity on the part of the cells in the tissues concerned, but the known difference in permeability of the capillaries in different regions should not be forgotten. That the swelling and œdema in inflammation consequent upon trauma are not wholly due to passive transudation, but to blood pressure, appears to be clear from extensive experiments directed to this point, more particularly in relation to cerebral injuries.² The malnutrition of the tissues which follows the infliction of the irritant, especially the deprivation of oxygen, results partly in chemical changes which increase the internal osmotic pressure; and this active force in the damaged tissues themselves is demonstrably greater than that of any blood-pressure, and is entirely independent of blood pressure in producing the œdema.

Clinical significance of dropsy.—Given a patient suffering from dropsy, the first endeavour should be to refer it to its cause; and since it can seldom, if ever, be ascribed to one single factor, whether state of blood, condition of blood pressure, or nutrition of the tissues concerned, the primary and predominant factor is to be sought for, which is then regarded as the cause, and becomes the object to which treatment is directed. The clinical states with which dropsy is associated are conveniently separable into those which lead to (1) a general and (2) a

¹ "Discussion on Pathology of Dropsy," Pathological Society of London, *The Lancet*, 1901, vol. i. p. 1012.

² See "Cerebral Pressure following Trauma," by W. B. Cannon, *American Journal of Physiology*, October 1901.

local œdema. To the former category belong certain heart states, some forms of renal disease and blood affections characterised by anæmia, and perhaps some toxæmias. Among local dropsies are the ascites connected with hepatic disorders, the œdema that depends upon the blocking of veins in limited areas, and that which results from inflammatory processes, both acute and chronic. The recognition of the local or general character of the swelling is important as indicating a cause of corresponding nature. But although some dropsies are general in the sense that all parts of the body are involved, it far from follows that the distribution is uniform; seldom, if ever, are all regions equally affected, and, speaking generally, the most dependent parts suffer the most. Nor is the distribution of the œdema bilaterally symmetrical whilst unequal in its vertical manifestation; various causes, such as clothing, position of the limbs, etc., will determine inequality on the two sides.

CARDIAC DROPSY

This, which is the commonest form of dropsy, may be looked at as the expression of failure on the part of the heart to compensate for the existing lesion of its valves, walls, or covering, whether this be associated with pulmonary disease or not, and for practical purposes may be taken as the first evidence of lack of adjustment; and, conversely, in a damaged heart compensation may be regarded as satisfactory so long as œdema is wanting.

The cardiac lesion with which the œdema is immediately connected is incompetence of the tricuspid valve, a slight degree of which, it is to be remembered, is readily induced—though probably temporarily only—within the limits of health. This incompetence may have followed on primary lung mischief, especially emphysema, or may have been secondary to disease at the mitral orifice, stenosis, or regurgitation, which has told back through the pulmonary circulation to the right side. Or the tricuspid insufficiency may have been caused by a dilatation of the ventricle consequent on myocardial degeneration. Thus it is that a febrile attack—rheumatic or other—may, by affecting the myocardium, or by causing pericarditis, determine an œdema in a case of cardiac disease in which hitherto the lesion had been adequately met. In congenital heart disease, however, even when severe, œdema is relatively rare, since in the mal-development of the heart satisfactory adjustments are for the most part established. Some curious differences, hitherto unexplained, are met with in the distribution of œdema as connected with the several heart lesions. Thus ascites is much more frequent in mitral stenosis than it is in mitral regurgitation, whilst general œdema of the integuments tends to be more marked in the latter. Whilst anasarca is commoner with a mitral regurgitant lesion, many cases of stenosis at this orifice exhibit no dropsy, but a very considerable engorgement of

the liver, which plays the part of a reservoir, allowing a large amount of blood to be held up, as it were, and relieving the engorgement of the right heart; and it is only after this condition has existed for some time that ascites sets in, the probability of this occurring being increased by the subsequent development of a tricuspid lesion. The incidence of some, perhaps slight, interference with the circulation, such as an unusual exertion, or a bronchial catarrh, enough to add a little to the difficulty of the pulmonary circulation, may be sufficient to determine a rapid increase in a hitherto slowly developing dropsy, whether of the integuments or of the serous cavities or of both. It is seldom that effusion into the pleural sacs is equal on the two sides, a singular preference being exhibited by the left, which may be alone, or almost alone, involved.

Disease of the aortic valves is seldom complicated by dropsy, unless there be coincident mitral disease or dilatation of the ventricle, rendering the mitral or tricuspid valves, or both, incompetent, to which condition is to be attributed any anasarca which may appear in the later stages as compensation fails.

Effusion into either of the serous sacs, peritoneum or pleura, very seldom occurs in heart disease independently of any general œdema, and the appearance of such a condition calls for inquiry as to the presence of some local complication.

Dropsy of cardiac or cardio-pulmonary origin commences, as a rule, in the feet, to which for some time it may be restricted. The influence of gravity in determining and maintaining œdema due to heart disease is very considerable, rest in the recumbent position being a most effective agent in its removal. In the distended veins the valves are rendered useless, and the weight of the column of blood contributes alike to the congestion and the œdema. From the feet the swelling spreads to the shins and higher parts of the legs and thighs, over the sacrum, and, when the case is advanced, extensively over the back and abdominal parietes, the trunk generally, and even the upper limbs and the face. Thus its degree is very various, and it is also very variable, shifting as it does with posture to the most dependent parts of the body, and changing with the state of the heart; disappearing when slight, after a night's rest, to reappear in the course of the day; more marked along the backs of the limbs in persons confined to the recumbent posture in bed; characteristically distributed over the sacrum in the orthopnoéal posture of urgent cardiac failure—"the sacral cushion." Œdema is recognised by the swollen, shining characters of the dropsical part and by pitting on pressure; in extreme degrees the integuments become tense, possibly red and tender, and may crack or give way.

A consideration of the conditions of occurrence and the character of dropsy associated with cardiac or cardio-pulmonary disease shows that the essential for its production is an impediment to the systemic venous return situated in the right heart, secondary to some obstruction

either in the pulmonary circulation or in the left heart. This would tend to raise the pressure on the venous side of the capillaries, and, provided the power of the left ventricle be maintained, the intra-capillary pressure would be increased. But the œdema is connected with a failure in the force of the left heart when the pressure falls in the systemic arteries and capillaries, and the blood flow is slower in the veins which are engorged. If to this be added some damage to the capillary wall as the result of malnutrition (for it has been previously stated that changes in blood pressure alone are insufficient to account for the transudation), then a condition exists which would favour the outward passage of fluid from the blood, at the same time hindering the absorption into the blood from the tissues; and consequently accumulation occurs. This is aided by the further obstruction to the drainage *via* the lymphatic vessels, which are compressed by the fluid with which the tissues are gorged, whilst the decreased aspiratory power of the heart, and probably also of the thoracic movements, still further interferes with the due removal of the increased effusion by these channels, as also with the return of the venous blood.

RENAL DROPSY

The association of general anasarca with certain forms of renal disease is well known. In chronic tubal nephritis—large white kidney—œdema is constant, and it is in such cases that the condition is seen at its greatest degree; moderate effusion into the serous cavities also occurs, but this does not become in the peritoneum so considerable as it is in hepatic cirrhosis. In acute nephritis and in lardaceous degeneration of the kidneys, anasarca is commonly, but not invariably, met with, some cases of these affections running their course with little or no œdema. A slight puffiness of the ankles and over the tibiæ may be noticed in the chronic interstitial form of nephritis, but there is no general œdema until the later stages, when a dropsy of cardiac determination supervenes from dilatation of the heart with failure of compensation.

Some relationship exists between the amount of urinary secretion and the extent of the œdema. This is best and most constantly marked in the chronic tubal nephritis, when the anasarca commonly increases with a diminution of urine, which is highly albuminous. In the other forms of kidney disease no such connection is observable, for although the urine may be suppressed in acute nephritis the occurrence of dropsy is not invariable; whilst a very large amount of urine may be passed in lardaceous disease with a moderate and persistent anasarca. The pallor of the integuments and the anæmia which are so pronounced in cases of this character offer a marked contrast to the congested and dusky or even cyanotic appearance of the patient whose dropsy is of cardiac origin.

Although the influence of gravity on the distribution of the œdema is not wholly wanting, it is not perhaps so marked as it is when a circulatory disturbance is the essential cause. Thus it is that the condition is not infrequently first noticed in the puffy eyelids where the subcutaneous tissue is lax and yielding, even before, or at least as soon as, it is perceptible in the lower extremities; and raising the legs is not so effective in relieving the swelling as it is in heart disease. Yet the position of the patient is apt to determine some inequalities in the distribution of the fluid.

In the causation of this form of dropsy the blood pressure is probably less effective than it is in the cardiac variety. For although the arterial tension is somewhat raised in all these diseases, in that, viz. chronic interstitial nephritis, in which it is highest the œdema is entirely or almost wholly absent until the pressure falls from a failing heart and the venous system becomes obstructed. But in these renal affections there exists a marked hydræmia, together with a blood containing an excess of effete and toxic substances, circumstances that especially favour an excessive transudation, partly from the quality of the blood and partly from an increased permeability of the vessels due to their mal-nutrition. A diminution in the absorption probably contributes less to the result than it does in the case of cardiac dropsy, where it forms an important factor. That the altered blood state is the chief agent in the production of renal œdema is confirmed by the decrease that occurs with an improvement in the quality of the blood, far more than takes place with any change that may be effected in the circulation, though it is not to be denied that in favourable cases, along with a general improvement in health, and an increase in the arterial tension and cardiac hypertrophy, the dropsy may almost or quite disappear. Dr. Dickinson (*Allbutt's System of Medicine*, vol. v. p. 687) has shown the great frequency with which acute renal dropsy is complicated by some inflammatory condition of the lungs, "often bronchitis or broncho-pneumonia. These conditions, though not the essential cause of the dropsy, but probably only connected with it as the result of a common cause, cannot but enhance the dropsical tendency." At the same time he points out that these pulmonary affections are "mostly acute and not such as to produce any considerable hypertrophy of the right ventricle."

The conditions under which œdema appears in renal disease, contrasting as they do with the states underlying cardiac dropsy, have suggested that the process is more akin to what obtains in inflammation, to be immediately considered.

ANÆMIC DROPSY

This form, although conceivably general, is much more commonly restricted to the ankles, feet, and legs, and is inconsiderable in amount.

Still, in severe cases of chlorosis or of hæmorrhage the œdema may be perceptible in the face, and to a very slight degree in the subcutaneous tissues generally.

In chlorosis, at least in the earlier stages, and until the debility becomes excessive, the pulse is full and of increased tension; but, as in renal dropsy, the probable explanation of the condition is to be found in the altered quality of the blood, which is favourable to transudation through a capillary wall whose permeability is increased from defective nutrition.

Unless the anæmia be very profound the œdema usually subsides after the lower limbs are raised to the level of the trunk. The restriction of the swelling to one limb should suggest the probability of thrombosis, which is a not infrequent result of the anæmic state (Vol. II. p. 334).

INFLAMMATORY DROPSY

In all vascular tissues the process of inflammation is accompanied by some œdema, the extent of which varies with the part affected and with the nature and severity of the cause. When the tissues are loose, or the serous sacs are the seat of the inflammation, then the effusion may be very considerable, as seen in facial erysipelas, in peritonitis, or in acute pleurisy. In the firmer and more resistant structures the amount poured out is necessarily less.

Inflammation being the response of the tissues to some abnormal irritation (associated with which is a vascular hyperæmia that subsequently tends towards stasis in the inflamed area), the tissue elements are primarily concerned, and hence the œdema may be regarded as in great measure dependent upon the perverted activity of these elements, any effect of altered blood pressure or quality of blood being subordinate. The œdema in fact is looked upon as being comparable to an excessive secretion on the part of the cells of the capillary walls and of the tissues. The altered activities of these cells resulting from the injury inflicted on them by the irritation lead to a breaking down of the big molecules into smaller and more diffusive ones. "There will in this case be an increase of the concentration of the intercellular fluid as compared with the blood, and fluid will be sucked from blood into tissue-spaces in order to equalise this difference of concentration. Such a process occurs in every secreting gland or contracting muscle, and must be partially responsible for the œdema produced by local injury or introduction of poisonous substances, *e.g.* the sting of bees. It cannot, however, be regarded as the main factor in the production of an enduring dropsy."¹

¹ Prof. Starling, F.R.S., "Pathology of Dropsy," *Trans. Path. Soc.*, Lond. 1901.

DROPSY OF LOCAL OBSTRUCTION

Such is the œdema which ensues upon thrombosis of venous trunks or pressure upon them by tumours, etc., or from varicose veins. One of the most frequently occurring and best marked illustrations of this group is the ascites associated with certain diseases of the liver.

HEPATIC DROPSY

The greatest accumulation of fluid in the peritoneal cavity is usually dependent on obstruction to the distribution of the portal blood in the liver as the result of cirrhosis. A similar ascites follows pressure on the trunk of the portal vein, though rarely to the same degree. The peritoneal effusion in cardiac obstructive disease, which immediately depends on the interference with the portal circulation, though often very considerable, is seldom so great in amount as in hepatic cirrhosis, nor does it recur as a rule so rapidly and so fully; the extent of ascites in renal disease has already been referred to as being but moderate.

It has been usual to explain the dropsy from obstruction of the portal vein, whether of the trunk or of its terminal branches in the liver, as being the direct result of the increased blood pressure in the portal area. It is very doubtful, however, whether this is the sole cause. For experimental ligature of the trunk of the portal vein has been performed without any ascites resulting (Starling), and in thrombosis of that vessel effusion into the peritoneal cavity is not an invariable sequence, at least not immediately. Moreover, very considerable cirrhosis, even of the atrophic form, may exist with but little or no ascites, and probably in most cases of this disease the change in the liver has proceeded to an advanced degree before effusion occurs, this being a later result. It should be remembered, however, that when an efficient collateral circulation is established through the anastomoses between the portal and systemic circulations at the junction of the coronary and œsophageal veins and elsewhere, the incidence of dropsy may be delayed. The rapidity with which the peritoneal fluid may re-collect after removal has also been regarded as against mere portal obstruction and increased blood pressure being the sole cause. No doubt, as in the œdema of cardiac disease, some change in the vessels of the portal area ensues upon the retardation of the blood flow, which facilitates the transudation; but there are certain considerations which would appear to suggest that the explanation of the ascites in portal obstruction is oftener to be found in some altered (inflammatory) state of the peritoneum. It is somewhat remarkable, if an increased blood pressure and altered capillary walls be the cause, that the effusion should take place into the peritoneal cavity, as it does, rather than into the lumen of the stomach and bowels, which it certainly does not; for it is on the mucous rather than on the peri-

toneal surface that the chief engorgement of the capillaries takes place, as is evident from the anatomical distribution of the vessels, and as is shown by the hæmorrhages which take place from the mucous membrane in such conditions.

That the determining cause of the ascites in many cases of hepatic disease is a chronic peritonitis appears to be most probable, especially in those which are characterised by perihepatitis. Dr. Hale White is of opinion that when the fluid re-accumulates and tapping is frequently repeated, this latter condition is always present, and that when the cirrhosis is uncomplicated by the capsulitis, ascites is a terminal phenomenon, and the patient succumbs after the first withdrawal and before a second is needed.

In hepatic cirrhosis, especially when the capsule is inflamed as a part of a chronic peritonitis, the vena cava is likely to be somewhat constricted in its fissure at the posterior part of the liver, and this may be partly responsible for the œdema of the lower limbs so commonly seen in extreme ascites.

DROPSIES OF UNDETERMINED CAUSE

Numerous cases of dropsy are met with that cannot be referred to either of the foregoing categories, and the nature of which is still obscure. The extent of œdema may range from a slight puffiness over the ankles and shins to a widespead anasarca with effusion into the pleural and other serous cavities.

Some are associated with definite nervous disease, as the combined neuritis and dropsy in varying proportion that characterise beri-beri (Vol. I. p. 353); or it may be that no nervous symptoms are present, and the œdema constitutes the chief feature, as occurs in the tropical malady known as epidemic dropsy (Vol. I. p. 360). The so-called angio-neurotic œdema (Vol. III. p. 384), which is frequently of a very localised distribution, is usually regarded as dependent upon some trophic disturbance of the blood-vessels or tissues of neurotic causation. The possibility, however, of all these conditions being really due to poisons acting as lymphagogues must not be overlooked; and a similar explanation may underlie the exceptional appearance of œdema in diabetes independent of renal disease or of cardiac failure, when the conditions would seem to be most unfavourable to transudation, and even in those rare instances when an extensive œdema occurs in persons apparently in otherwise perfect health, to which they return as the dropsy subsides.

Nature of the fluids effused.—Not only do the capillaries in various regions of the body differ in their degree of permeability, as stated above, but they also differ in the readiness with which they permit the several ingredients of the blood plasma to transude. Thus the

hepatic capillaries are more permeable than those of the intestines, and still more than those of the limbs; and though the amount of salts found in the effusions from these several sources will be about the same, and closely correspond to what is found in the blood, the proteids in the lymph from the liver capillaries will much exceed that from the limbs, that from the intestines being intermediate in amount. Thus it is that the dropsical fluids from the peritoneum, from the pleural cavities, and from the subcutaneous tissue differ considerably in composition, quite apart from the disease to which the dropsy is due. These differences especially affect the proportion of the contained proteids, these being most abundant in pleural and pericardial effusions, least in subcutaneous œdema, and intermediate in quantity in ascitic fluid. In all cases the inflammatory effusions are much richer in proteids than those which have resulted from venous obstruction, anæmia, or renal disease. The specific gravity of the latter also is usually lower than when inflammation is the cause, being 1010 to 1015, as compared with 1018 or higher. The coagulability of the inflammatory exudations is also much greater than that of the simpler transudations.

These various fluids are all alkaline in reaction and closely resemble each other in their composition. The proteids are serum-globulin, serum-albumin, and extremely small quantities of fibrinogen. Sugar is usually present, and occasionally cholesterol. The salts are almost identical with those of the blood, the chlorides being most abundant. Of 77 cases of effusions of different kinds, Runeberg found the total amount of proteids ranged from .06 to 2.68 per cent, while the salts scarcely varied throughout the series. The tint of these fluids is usually some shade of straw colour, pale or dark; they may be bile-stained when jaundice is present, or blood-stained. This last is commonly the case when the effusion is associated with the existence of a new growth, but not necessarily so, for the same may occur from the rupture of the new-formed capillaries developing in the fibrinous adhesions which are becoming organised. It is noteworthy that the fluid removed by successive tapplings from the same cavity exhibits a marked constancy in composition, unless inflammation should supervene, and then the relative proportion of proteids is much increased.¹

The effusions met with in the serous cavities, more particularly the abdominal, are sometimes *milky* in appearance. Although collectively designated as *chylous*, they are by no means always of that nature. The fluid is opalescent, milky, or even quite white, of somewhat variable composition. True chylous ascites, when the fluid is really chyle, is due to escape from the thoracic duct or abdominal lymphatics which are obstructed with rupture, or to invasion of the vessels by tuberculosis or malignant growths; or it has been supposed that the chyle may transude through

¹ For further details reference may be made to Professor Halliburton's *Text Book of Chemical Physiology and Pathology*.

diseased lacteals without actual rupture. In other cases the appearance of chyle may apparently be simulated by the presence in the fluid of certain peculiar proteid substances, of which a casein-like body—glycoproteid—has been recognised. An abundance of cholesterol crystals has been said to give a tinted, somewhat opalescent appearance, and a variable quantity of fat granules is often present. This condition has been recorded as being associated with many different abdominal affections, of which cirrhosis of the liver, tuberculosis, and malignant disease of the peritoneum are the most usual. Similar fluids have been found in the pleural sacs.¹

Physical signs and effects of dropsy.—The effusion of fluid into the tissue spaces or into the serous sacs will produce more or less swelling, according to the quantity poured out and the degree of resistance offered by the structures involved. The effects of œdema of a limb are to cause it to increase in size, with filling up of the depressions and disappearance of the prominences, thus tending to render the part of uniform size, and by the mechanical impediment to interfere with free movement of the joints. In such situations as the scrotum, eyelids, face and neck, the extent of swelling may be enormous from the laxity of attachment of the skin to the deeper parts. The skin, being stretched, becomes smooth and shiny as the normal wrinkles are obliterated. On pressing the surface with the finger a depression is caused, known as “pitting,” which may last for some time; when very slight in amount this condition may be better felt than seen by passing the finger over the depressed area. The constriction of garments, garters, etc., may cause a deep grooving, the swollen integuments rising up on each side, or more markedly on the distal. The colour of the integuments will vary very much with the cause of the œdema, being pallid and waxy in anæmia and in renal disease, of a bright red when the skin is inflamed, as in erysipelas, or purplish and cyanosed, with distended venules, when the swelling is the result of venous obstruction. Accumulation in the serous sacs also causes increase in size with alteration in shape, most marked in the abdomen, and much less when the pleural or pericardial cavities are the seat of the effusion, the rigid chest-wall, especially in adults, yielding but little. In ascites the enlargement is uniform, unless any other cause of distension, *e.g.* new growth, should co-exist, with some bulging of the flanks when the patient is on his back, though the extent to which this sign is appreciable depends very considerably upon the looseness or resistance of the parietes, being much more marked when the elasticity of the integuments is diminished and the muscles are soft, and with but little subcutaneous fat. As the abdomen distends, the umbilicus either becomes retracted from the drag of the remains of the attached foetal structures, or, as oftener occurs, the

¹ For an account of recorded cases, see paper by Dr. Batty Shaw, *Journal of Pathology and Bacteriology*, Feb. 1900.

folds of the navel are flattened out. The maximum girth of the abdomen is at or above the umbilicus, and the measurement from that point to the xiphoid cartilage exceeds that to the pubes. Due to the stretching of the skin, "lineæ albicantes" are formed over the lower part of the abdomen, and frequently extend to the outer and upper part of the thighs.

As the fluid collects and displaces the lungs, intestines, etc., a *dull note* is obtained on *percussion* in place of the characteristic resonant or tympanitic sound elicited in the normal state, and thus the whole side of the chest (excepting perhaps the upper and inner part posteriorly, to which situation the compressed lung is driven) or the entire abdomen may be dull. When the fluid has not reached so great an amount the epigastric and part of the adjacent regions may be tympanitic, the line of the dull area taking a curved direction with the concavity upwards. The relative extent of tympanitic and dull areas in ascites will be conditioned by the degree of abdominal distension, by the length of the intestinal mesenteries, by the existence of adhesions between the viscera, and also by the presence of solid tumours. Should the amount of fluid in the cavity be but small, it may be necessary to place the patient on his hands and knees to allow it to gravitate to the most dependent part, over which a dull note will be obtained on percussion. A most characteristic feature of the *limits of dulness* is that they *shift with change in the position* of the patient from side to side, or from the recumbent to the erect posture; this is far more readily to be recognised in ascites than in pleuritic or pericardial effusion, when it is often quite inappreciable.

When the amount of ascites is at all considerable a peculiar sensation, known as *fluctuation*, is to be felt when the hand is applied over one side of the abdomen and the opposite side is tapped with the other hand. Care must be taken to distinguish this from the vibrations communicated under the same conditions by a very tense abdominal wall. Should the fluid be encysted this sign may not be elicited, and it is very seldom to be appreciated over the intercostal spaces in pleuritic effusion.

The special auscultatory phenomena determined by pleural or pericardial effusion are detailed elsewhere (pp. 257 and 426).

The collection of fluid in the serous cavities is necessarily productive of more or less displacement of adjacent viscera and often some impairment of their function. Thus it is that as the pleural cavity becomes filled, the lung, unless fixed by adhesions, is displaced upwards, backwards, and inwards, until in a state of collapse from compression it lies close along the spine in the upper part of the thorax. Dyspnoea to a varying extent is the result, and the more so the more rapid the effusion. Displacement of the heart to the right or left—much greater in the former case—will follow according to the side of the effusion, and this may determine all degrees of disturbed cardiac action even to syncope. Similarly a pericardial effusion will, in proportion to its extent, impede

the heart's action, and cause a largely increased area of percussion dulness, extending even to the back, with some displacement of the left lung as a consequence. Nor are the effects of these intrathoracic effusions limited to the chest; when extensive they are usually accompanied by depression of the liver, and to some extent of the stomach, this being most obvious when the left pleura is the site of the accumulation.

Due probably to the more yielding character of the abdominal walls, the pressure effects of ascites are scarcely so constant or so obvious. Yet when the dropsy is very great the heart may be displaced considerably upwards and outwards, giving rise to irregularity of beat, to palpitation and faintness, and sometimes even causing a basic systolic bruit. By interfering with the action of the diaphragm and the proper expansion of the lower part of the lungs, a congestion of these organs is favoured, leading to some difficulty of breathing and increased movements of the upper part of the chest. It is the pressure effect upon the heart and lungs that renders any extreme condition of ascites one of gravity, the patient often passing rapidly into a state of danger and urgently requiring paracentesis to save his life. The stomach and intestines undergo some amount of compression, but this is rarely sufficient to be of practical importance. The uterus may be forced downwards, even to protrusion of the os at the vulva. The pressure also frequently affects the large venous trunks, especially the renal, as shown by a slight albuminuria as a result of the congestion of the kidney, and the vena cava, causing some œdema of the lower extremities, a condition which may also be due to compression of this vessel from fibrosis as it passes behind the liver, as already referred to.

The bulk of ascitic fluid gives rise to a sense of weight and of dragging pain in the loins, seriously interfering with the patient getting about. A similar feeling of oppression accompanies the pleural accumulation, and when the pericardium is distended considerable distress is manifest. Rupture of the abdominal wall or a giving way at the umbilicus is not unknown when the distension has been unusually great and the parietes are wasted and thinned.

Prognosis.—This is necessarily dependent on the cause. While in many cases it is distinctly relieved by treatment, it tends to recur in a large proportion of cases, and in the final stages of obstructive heart disease contributes in a great measure to the fatal ending. On the other hand, many cases of ascites, even after repeated tapping, will undergo a spontaneous cure.

Treatment.—This should of course be directed when possible to removing or remedying the cause, and in such conditions as the œdema of anæmia this may be efficiently carried out, and nothing more is required. Most frequently, however, this is impossible. Most cases of chronic cardiac obstruction, of renal disease, or of hepatic cirrhosis are

incurable, though much may be done to alleviate symptoms and to prolong life ; hence attempts to reduce the dropsy should be made, as this state adds much to the patient's discomfort, and may of itself seriously increase the risks of the primary affection. Much the same may be said in regard to the local œdema due to the pressure of tumours on large veins.

The influence of gravity in the production of œdema has already been referred to, and from this it follows that the horizontal posture becomes an important factor in the treatment of this condition, rendering more efficacious any other means that may be employed. When the dropsy is limited to the lower extremities it may subside almost or quite by a few days' rest in bed ; and when of more extensive distribution it may compel this course, owing to the physical difficulty in getting about. Should the patient be orthopnœic, such a plan can only be partially followed, and no doubt a long maintained recumbent position does favour anasarca of the back and across the loins, as well as œdema of the lungs, all of which may be in some degree averted by directing the patient to lie periodically on one or other side, or even on the face. Where, however, the primary cause is cardiac obstruction, much difficulty is often experienced in pursuing this treatment, the patient's restlessness and desire to sit up out of bed defying such recommendations. In such cases it is well to allow him to pass his time in a chair, so long as he feels inclined. As supplementing rest, much benefit may follow careful bandaging of the limbs ; the uniform pressure thereby exerted and the support given to the superficial veins assist the circulation and aid in the absorption of the effusion.

The principles of the treatment of dropsy are based on measures to promote the absorption of the fluid and its removal by the normal excretory channels ; on the withdrawal of the effusion by tapping, puncture, etc. ; and so far as possible on the prevention of further transudation. Owing to the great variety of causes of dropsy, the extent to which either of these plans is applicable in any given case will necessarily vary, and will much depend upon the condition of the circulation, the quality of the blood, and whether the dropsy be general or local.

To promote the absorption of the fluid where the œdema is general and is due to central (cardiac) obstruction, it is of fundamental importance to restore as far as possible the impaired circulation ; and as this is in a great degree connected with engorgement of the venous system, and determines in a large measure both the extent of the effusion and the power of absorption, whatever further treatment is adopted, it is frequently well to begin with a withdrawal of blood, whether by cupping or leeches over the liver, over the lungs, or over the heart, (which on the whole is generally preferable to venesection in the arm), to the extent of a few ounces (5 to 8) of blood, a procedure that of itself

will usually improve the vascular tone and lead to absorption of the effusion. This course is especially beneficial in dropsy of cardiac or cardio-pulmonary origin, but it is rarely advisable in renal dropsy, unless it be in the later stages, when heart failure is supervening. Most valuable agents in this mode of treatment, as supplementary to the all-important rest in bed, are such drugs as digitalis, strophanthus, and citrate of caffeine. The form in which they may be given is variously recommended, but no one is more frequently applicable than the well-known pill containing a grain of each of the following :—Pulv. digitalis, pulv. scillæ, and pil. hydrargyri, a combination in which the tonic action of the digitalis on the cardio-vascular musculature is supplemented by the diuretic action of the squill and the relief of the portal congestion by the blue pill. Concerning the administration of digitalis for the improvement of cardiac power and vascular tone, it may not be inappropriate to point out that this drug has certain advantages over others of its class, insomuch as it increases the tonic activity of the arterial musculature, as well as that of the myocardium, whilst this latter property alone is characteristic of other drugs of the group of so-called “cardio-vascular tonics.” For this reason digitalis is the most valuable in the condition under consideration, viz. œdema and dropsy, associated with an enfeebled heart and an engorged venous system, and its place is very imperfectly taken by strophanthus, squill, convallaria, and others ; although in exceptional cases, for reasons not understood, they may succeed, where digitalis has failed, in improving the cardiac power. At the same time it is to be remembered that toxic symptoms are easily produced by those drugs, especially nausea, vomiting, diarrhœa, and a liability to syncope, and that with excessive doses the secretion of urine is diminished and the consequent elimination of their active principles. It is usual also to refer to what is known as the cumulative action of these substances, and to give a warning as to their moderate administration on that account ; but, however true this may be when digitalis is studied experimentally in animals, the writer is fully in accord with those who are inclined to regard the circumstance as of no practical importance under the ordinary therapeutical conditions in which they are prescribed. For this reason it appears to be not only desirable but justifiable that the drugs should be given in sufficiently full doses to produce the desired effect ; and whilst five or ten drops of the tincture of digitalis or strophanthus are commonly given three or four times in the twenty-four hours, this amount may with great advantage be doubled or even trebled should occasion require, the administration being stopped directly the full effect is obtained. Of the relative merits of the several pharmacopœial preparations it is difficult to speak with certainty, as much depends on the recency of their manufacture, but the efficacy of an infusion of the freshly gathered leaves of the foxglove is undoubted.

Inasmuch as massage, when properly performed, accelerates the flow of blood and lymph, and thus aids in the absorption and removal of fluid effusions, it may under certain circumstances be beneficial in œdema, more particularly when this is localised. In the anasarca of chronic Bright's disease, or of cardiac obstruction, it requires the greatest care in application, and in accumulations in the serous cavities it is of no avail. The benefit which follows is probably due as much to improved metabolism as it is to the actual assistance afforded to the circulation.

Next to facilitating the absorption of the effusions by an improvement in the circulation, which at the same time, be it observed, tends to diminish the production of the œdema, the object is to effect the removal of the fluid by the several excretory channels, kidney, bowels, and skin. The most efficient diuretics are the digitalis group of cardiac tonics, which act by raising the renal blood pressure, the blood-vessels being constricted at the same time that the rate of blood-flow through the kidney is increased. The consequent augmentation of the flow of urinary water, the solids being but slightly affected, is to be regarded rather as a result of over-secretory activity on the part of the renal cells than of simply increased filtration, since the condition of the circulation in the organ would cause a fall of the pressure in the glomerulus where the water is secreted, a condition unfavourable to an increase of flow. Caffeine, which is one of the most powerful of diuretics, produces this effect by a dilatation of the renal vessels, this being most marked with the first doses, a contrary result tending to ensue as the drug subsequently causes constriction of the renal vessels. Hence, like digitalis and its allies, caffeine and theobromine, which is similar in action, are most useful in the dropsies of cardiac origin, having less effect on those associated with renal disease, and still less with the ascites due to portal obstruction. The direct stimulant action of the last-named drugs on the renal epithelium necessitates their administration with care in structural disease of the kidneys. Although digitalis and caffeine have such an opposite effect upon the vessels of the kidney, they are often prescribed in combination with advantage in cardiac dropsy, the effect of the former on the heart and of the latter directly upon the kidneys probably explaining the result (*vide* Dr. Bradford in *Text-book of Pharmacology and Therapeutics*, edited by Dr. Hale White, 1901).

Infusum scoparii or infusum uvæ ursi, from their slight diuretic action, are frequently used as excipients. For ascites of hepatic origin the resin of copaiba is often most effective, though it is very uncertain in its action. Combined with digitalis it has been given in cardiac dropsy, but its use is to be deprecated in the anasarca of renal origin. A convenient formula for its administration is the following, to be given thrice daily :—

not only in reference to the causal state, but also in its direct bearing on the diminution of the swelling. It might appear that a restriction of the intake of fluid would affect the amount of effusion, but as a therapeutic measure it is seldom convenient or even very effective, and in renal dropsy is not to be recommended, tending as such a course does to prevent the proper elimination of the solid waste. The same objection does not hold in respect to the effusions of cardio-pulmonary disease, of hepatic cirrhosis, or of pleurisy, pericarditis, or peritonitis, in all of which the method is sometimes efficacious and may even be so without other measures; but at least it must be regarded as uncertain, and is probably most useful in conjunction with other remedies. Large quantities of skim-milk, several pints daily, have been found to be an effective diuretic, and for a time renal anasarca may be so treated; an increased elimination of the nitrogenous and other urinary solids and some decrease in the albuminuria being the result. At the same time, however, the value of a more liberal and nourishing diet in the dropsy of kidney disease is often most marked, notwithstanding what is often regarded, though with doubtful propriety in all cases, as the necessity for reducing the nitrogenous aliment to a minimum. As an accessory to more direct methods of removing the fluid, diaphoretics, puncture, etc., such a regimen is of great benefit, provided that the urinary secretion be not diminished. The anæmia that is usually associated with chronic tubular nephritis also requires attention.

All such measures as tend to improve the general health and tone, such as fresh air, cleansing of the skin by tepid or warm sponging, mental quiet and acceptable surroundings, may contribute to the amelioration of anasarca from any cause by favouring the action of the more specific modes of treatment above set forth. It is, moreover, desirable that the condition should be attacked simultaneously in several of the directions indicated, as thereby a greater chance of improvement is likely to occur.

The radical treatment, with consequent relief and sometimes even cure, of œdema or dropsy is the direct removal of the fluid by puncture or by tapping. The subcutaneous effusion is rapidly drained away by a few punctures over the legs, which may be permitted to hang down as the patient assumes a sitting posture. Care should be of course taken that the instrument—a hare-lip needle is the most convenient—be absolutely clean, that the skin have been previously washed with an antiseptic solution, and during the drainage that all risk of infection be avoided. With such precautions the œdema from any cause may be so treated, but it should be remembered that sloughing is more likely to follow when the kidneys are diseased. The scrotum may be so punctured and with much relief, but it is rarely advisable to follow the same course with the penis, however great the swelling. Many pints of serous fluid may be removed in this manner, at the same time that

other remedies are being administered. The insertion of Southey's tubes beneath the skin of the legs is an efficient and cleanly method of attaining the same result.

Accumulations in the serous cavities may require paracentesis, and the necessity for, as well as the method of, the procedure in respect to the pleura is discussed on p. 264. The propriety of tapping for ascites is largely conditioned by the cause of the dropsy; for whilst seldom required in renal disease, it may become a matter of urgent necessity in cardiac disease, in hepatic cirrhosis, or in chronic peritonitis. The incision for the trocar is usually made in the middle line about midway between the pubes and umbilicus, care being taken to have the bladder empty, and the fluid drained off into a pail, the end of the tube being so disposed as to prevent the entrance of air. It is sometimes desirable to effect the removal of the fluid very slowly, so as to avoid the risk of too great a distension of the abdominal blood-vessels with consequent fall of pressure, which might be fatal. This may be carried out by using a Southey's tube for the purpose, twenty-four hours or so being occupied in the procedure. In all circumstances it is desirable to keep up pressure by a constantly adjusted "many-tailed bandage" as the fluid drains away; and all antiseptic precautions with respect to the operation should be observed. Paracentesis abdominis may be repeated again and again—it has been performed over 150 times in the same patient; and in a certain proportion of cases reaccumulation ceases to occur, and so far as that condition is concerned a cure may be effected. This subject will be further discussed in connection with Diseases of the Liver and Peritonitis (see Vol. V.).

Another method of treatment of ascites consequent upon hepatic disease, especially perihepatitis, which has been recently recommended, and for which some success is claimed, consists in opening and draining the abdominal cavity and then attaching the omentum to the abdominal wall, whereby adhesions are set up. The object aimed at by this procedure is to establish a collateral circulation between the mesenteric and parietal vessels, and so relieve the congestion due to the portal obstruction. Whether this is really effected to any appreciable extent is uncertain, or whether such improvement as does sometimes follow is the result of any improvement in the nutrition of the liver which is thereby established, or whether it be from a closure of the peritoneal cavity by setting up universal adhesions, is equally unknown.

W. H. ALLCHIN.

INDEX

- Abscess, pulmonary, 72, 209
- Accentuation of cardiac sounds, 345
- Actinomycosis, cardiac, 335
 - pulmonary, 97
- Adenoids, 33
 - etiology, 34
 - morbid anatomy and pathology, 35
 - symptomatology, 35
 - treatment, 39
- Adherent pericardium, 331
- Adventitious sounds in chest, 134
- Egophony, 133
- Age in cardiac disease, 321
 - pulmonary disease, 50
- "Air hunger," 113
- Air, respiratory changes in, 15
 - impurities in, causing disease, 53
- Alveoli, pulmonary, structure of, 6
- Aneurysm, aortic, 450
 - cardiac, 327, 417
- Angina pectoris, 368
 - false, 370
- Aorta, coarctation of, 337
- Aortic diastolic murmur, 350
 - incompetence, 404
 - obstruction, 403
 - orifice, congenital affections of, 337
 - systolic murmur, 348
- Apnoea, 21
- Arterial sounds and murmurs, 355
 - system, 304
- Arteries, diseases of, 438
 - embolism of, 440, 447
 - general etiology of, 438
 - morbid anatomy and pathology of, 439
 - thrombosis of, 439
- Arterio-capillary fibrosis, 441
- Arteritis, 440, 443, 459
 - acute, 444
 - chronic, 444
- Arteritis obliterans, 441
 - syphilitic, 441
- Asphyxia, 22
- Asthma, pulmonary, 50, 175
 - pathology, 176
 - symptoms, 177
 - treatment, 180
- cardiac, 372
- hay, 40
- Atelectasis, 85
- Atheroma, 326, 442, 448
- Auscultation of cough, 136
 - of heart, 344
 - of lungs, 130
- Auscultatory percussion, 136
- Barking cough of puberty, 104, 152
- Blood, gases of, 17
- Blood-pressure, intra-cardiac, 299
- Blood-vessels, diseases of, 438
- Bronchi, anatomy of, 6
 - dilatation of, 77, 170
- Bronchiectasis, morbid anatomy and
 - pathology, 77
 - symptoms, 170
 - treatment, 172
- Bronchiolectasis, 80
- Bronchiolitis, 177
- Bronchitis, acute, morbid anatomy of, 61
 - symptoms of, 155
 - treatment, 159
- capillary, 65, 161
- Bronchitis, chronic, morbid anatomy, 62
 - symptoms, 162
 - treatment, 164
 - plastic or fibrinous, 63, 167
- Broncho-pneumonia, diagnosis, 193
 - morbid anatomy, 64
 - symptoms, 190
 - treatment, 194

- Bruit d'airain, 136
 de drapeau, 168
 de galop, 345, 371
- Capillary circulation, 308
 pulse, 304, 356
- Cardiac acceleration, 363
 aneurysm, 327, 417
 apex beat, 298, 339, 340
 arrhythmia, 296
 contraction, 295
 dilatation, 332, 379, 419
 excitement, 361
 failure, 370, 378, 385, 391
 hypertrophy, 331, 379, 419
 impulse, 298, 339, 340
 infrequency, 364
 intermittence, 365
 irregularity, 365
 muscle, 294
 parasites, 335, 423
 strain, 423
 syphilis, 325, 335, 418
 weakness, 361
- Cardio-pulmonary murmurs, 137
- Casts, bronchial, 63, 167, 178
- Catarrh, acute nasal, 25
 etiology and pathology, 25
 morbid anatomy, 27
 symptoms, 27
 treatment, 28
 chronic nasal, 46
- Cerebral circulation, 313
- Charcot-Leyden crystals, 168, 178
- Chest, shape of, 123
- Cheyne-Stokes breathing, 115
- Chylothorax, 255, 272
- Circulation, general physiology of the, 288
 influence of gravity in the, 305
- Climate in pulmonary disease, 55
- Clubbing of fingers and toes, 117
- Coarctation of aorta, 337
- Cold and chill, 54
- Collapse, pulmonary, morbid anatomy, 85
 symptoms, 195
- Compensation in heart disease, 378
 failure of, 383
- Congenital affections of heart, 335
 diagnosis of, 435
 symptoms of, 433
- Congestion, pulmonary, 66, 74, 186
 hypostatic, 74, 187
- Coronary circulation, 315
- Coryza, acute. *See* Catarrh, acute nasal, 25
- Cough, 14, 102, 373
- Course and prognosis, general, of heart disease, 376
- Curschmann's spirals, 168
- Cyanosis, 116, 434
- Degeneration of heart, 323
 fatty, 318, 323, 417
- Diaphragm, disorders of, 283
 paralysis of, 286
 spasm of, 284
- Dilatation, bronchial, 77, 170
 cardiac, 332, 379, 385, 419
 "Dittrich's plugs," 170
- Dropsy, 464
 anæmic, 475
 cardiac, 375, 472
 clinical significance of, 471
 hepatic, 477
 inflammatory, 476
 physical signs and effects of, 480
 renal, 474
 treatment, 482
- Dry pleurisy, 260
- Ductus arteriosus, patent, 337
- Dyspnœa, 21, 110
 cardiac, 372
- Embolism, pulmonary, 76, 188, 458
 air, 77
 fat, 76
- Emphysema, acute vesicular, 82
 atrophous, 84
 chronic general hypertrophous, 82
 local hypertrophous, 83
 interlobular, 84
 morbid anatomy, 82-84
 production of, 80
 prognosis, 185
 symptoms, 183
 treatment, 185
- Empyema, 253, 259
- End-arteritis obliterans, 441
- Endocarditis, acute, 327, 396
 chronic, 329, 398
- Epistaxis, 29
 etiology, 30
 treatment, 32
- Etiology, general, of heart disease, 317
 of pulmonary disease, 50
- Eupnœa, 21
- Expectoration. *See* Sputum, 104
- Expiration, physiology of, 10
- Fibrosis, pulmonary, 90
 cardiac, 324, 416
- Fœtid bronchitis, 163
- Foreign bodies in the air passages, 54, 174

- Fremitus, bronchial, tussive, vocal, 127
pericardial friction, 342, 351
pleural friction, 127
- Gangrene, causes, 71
morbid anatomy, 71
symptoms, 210
treatment, 212
- Gases, of blood, 17
respiratory exchange of, 15, 19
- Hæmopericardium, 429
- Hæmoptysis, 108
- Hæmothorax, 255, 272
- Harrison's sulcus, 124
- Hay asthma. *See* Hay fever, 40
- Hay fever, 40
etiology and pathology, 41
prognosis, 43
symptoms, 43
treatment, 44
- Heart. *See also* Cardiac
anatomy of, 293
contraction of, 295
degenerations of, 323, 417
diseases of, 317
disorders of associated organs in heart
disease, 372
vascular supply of, 323
disturbances of force of, 361, 368
rate of, 363, 368
sensibility of, 366, 368
fibrosis of, 324, 416
functional disorders of, 430
inflammation of, 326
innervation of, 296
morbid anatomy of, 323
sounds of, normal, 301
changes in, 345
tone of, 295
valves, chronic disease of, 398
work of, 301
- Hepatisation, red and gray, 67, 68
white, 88
- Heredity in pulmonary disease, 51
- Hydatid of heart, 335, 423
lung, 96, 246
pleura, 273
- Hydropericardium, 323, 429
- Hydrothorax, 255, 271
- Hyperpnoea, 21
- Hypertrophy, cardiac, 331, 379, 419
- Hypostasis, 305
- Hypostatic pulmonary congestion, 74, 187
pneumonia, 187
- Infarction, pulmonary, 75
- Infections, as causing cardiac disease, 317
pleural disease, 251
pulmonary disease, 56
- Inspiration, physiology of, 9
- Interrupted respiration, 21
- Invasion, channels of, in tuberculosis, 57
- Laryngeal growths, 148
neuroses, 151
paralysis, 153
spasm, 152
stenosis, 144
tuberculosis, 145
vertigo, 152
- Laryngitis, morbid anatomy of, 60
sicca, 60
symptoms and laryngoscopic appearances, 139, 141
treatment, 140
- Larynx, anæmia of, 73
anatomy of, 2
hyperæmia of, 73
muscles of, action of, 3
neuroses of, 151
nerves of, 3
new growths of, 97, 148
œdema of, morbid anatomy, 72
symptoms and treatment, 140
perichondritis of, 61, 143
stenosis, 84, 144
syphilis, 87, 144
tubercle, 89, 145
- Lungs. *See also* Pulmonary
anatomy of, 4
new growths of, 98
structure of, 7
vessels of, 7
- Lymph, normal production and removal of, 464
- Lymphagogues, 468
- Lymphatic vessels, flow in, relation of to blood-vessels, 309, 310
- Malformations of heart, 335
- Mediastinal lymphatic glands, diseases of, 277
- Mediastinitis, acute, 274
chronic, 274
- Mediastinum, diseases of, 274
new growths of, 277
- Mitral incompetence, 400
presystolic murmurs, 351
stenosis, 401
systolic murmur, 349

- Murmurs, cardiac, 301
 cardio-pulmonary, 137
 endocardial, 346
 functional, 348
 hæmic, 302, 348
 pericardial, 351
 Myocarditis, 326, 415
 chronic, 416
- Nasal catarrh, acute, 25
 chronic, 46
 mucous membrane, special affections
 of, 46
 syphilis, 48
 tuberculosis, 47
- New growths of heart, 334, 422
 larynx, 97, 148
 lung, 98, 243
 mediastinum, 277
 pleura, 272
- Occupation in heart disease, 321
 pulmonary disease, 53
- Edema, 464
 laryngeal, 72, 140
 pathology of, 468
 pulmonary, 75
- Pachydermia laryngis, 61, 142
- Pain in diseases of heart, 366
 respiratory organs, 118
 referred, pulmonary, 119
 cardiac, 367
- Palpable diastolic shock, 342
- Palpitation, 368
- Pectoriloquy, 133
- Percussion of heart, 343
 lungs, 128
 resistance, 130
- Pericarditis, acute, 330, 425
 chronic, 427
 tuberculosis, 429
- Pericardium, 294
 adherent, 428
 defects of, 337
 diseases of, 425
 white patches on, 331, 429
- Pharyngeal tonsil, hypertrophy of, 33
- Phlebotaxis laryngis, 73
- Phlebitis, 461
- Phthisis, acute pneumonic, 91, 217
 chronic, morbid anatomy, 92
 symptoms, 219
 physical signs, 225
 diagnosis, 228
 prognosis, 230
 treatment, 233
- Physical examination of chest, 123
 heart, 339
 lungs, 123
 vessels, 352
- Pigeon breast, 124
- Pleura, arrangement of, 5
 diseases of, etiology, 250
 morbid anatomy, 252
 hydatid of, 273
 morbid growths of, 272
 tuberculosis of, 272
- Pleurisy, acute, etiology, 251
 morbid anatomy, 252
 physical signs, 257
 symptoms, 256
 treatment, 263
 chronic, 260
 diaphragmatic, 258
 dry, 260
- Pneumococcus, 56
- Pneumoconiosis, 70
- Pneumonia, acute, 65, 196
 complications, 202
 morbid anatomy, 65
 prognosis, 206
 symptoms, 196
 varieties, 203
 treatment, 207
 chronic, morbid anatomy, 69
 symptoms, 212
 hypostatic, 74, 187
- Pneumothorax, 254, 268
- Portal circulation, 314
- Præcordia, area of dulness of, 343
 distress over, 368
 prominence of, 339
 thrill over, 342
- Prognosis of heart disease, 376
- Pulmonary abscess, 72, 209
 apoplexy, 75
 circulation, 312
 collapse, 85, 195
 congestion, 66, 74, 186
 embolism, 76, 188
 fibrosis, 93
 gangrene, 71, 210
 growths, 98, 243
 œdema, 73, 186
 syphilis, 87, 248
 thrombosis, 77, 188
 tuberculosis, acute, 90, 214
 chronic, 92, 219
- Pulmonary artery, diseases of, 457
- Pulmonic diastolic murmurs, 350
 incompetence and obstruction, 406
 orifice, abnormalities of, 336

- Pulmonic systolic murmur, 349
 Pulse, arterial, 297, 302, 352
 capillary, 304, 356
 venous, 304, 357
 Pulsus bigeminus, 366
 paradoxus, 116
 Pyo-pneumo-pericarditis, 331, 430
 Pyo-pneumothorax, 268
 Pyrexia in affections of respiratory organs,
 101

 Radioscopy in diseases of chest, 138
 Râles, 135
 Reduplication of cardiac sounds, 345
 Respiration, cutaneous, 19
 extraordinary, 11
 gaseous exchange in, 15, 19
 interrupted, 21
 mechanism of, 8
 nervous, 20
 sounds of, normal, 12
 Respiratory organs, anatomy of, 1-8
 quotient, 16
 tract, lower, diseases of, 50
 upper, disorders of, 25-50
 Rhinitis, acute. *See* Catarrh, acute
 nasal, 25
 Rhonchi, 134
 Rose cold. *See* Hay fever, 40
 Rupture of heart, 333, 422

 Septa of heart, abnormalities of, 336
 Sex, in cardiac disease, 321
 pulmonary disease, 51
 Shock sound, 346
 Singers' node, 61
 Skodaic resonance, 129
 Sneezing, 14
 Spacing of cardiac sounds, 346
 Sputum, characters of, 104
 Stenosis, bronchial, 85, 173
 laryngeal, 84, 144
 tracheal, 85, 173
 Strain, cardiac, 423
 Stridor, 133
 congenital, of infants, 154
 Succussion sound, 137
 Symptomatology, general, of cardiac
 disease, 360
 of pulmonary disease, 101
 Syncope, 363
 Syphilis, cardiac, 335, 418

 Syphilis, laryngeal, 87, 144
 nasal, 48
 pulmonary, 87, 248
 tracheal, 87, 248

 Tenderness, referred cardiac, 343
 pulmonary, 122
 Thrombosis, cardiac, 333
 pulmonary, 77, 188
 venous, 460
 Tight lacing, effects of, on chest, 125
 Trachea, anatomy of, 5
 inflammation of, 61, 62
 stenosis, 85
 syphilis of, 87
 tubercle of, 96
 Tracheitis, acute, morbid anatomy, 61
 symptoms, 155
 treatment, 159
 chronic, morbid anatomy, 62
 Trachoma, 61
 "Traube's plugs," 170
 Traube's space, 129
 Treatment, principles of, in heart disease,
 389
 Tricuspid incompetence, 405
 obstruction, 406
 presystolic murmurs, 351
 systolic murmurs, 350
 Tubercle bacillus, 57
 Tuberculosis, arrested pulmonary, 96
 cardiac, 335
 laryngeal, 89, 145
 nasal, 47
 pleura, 272
 pulmonary, acute miliary, 90, 214
 chronic miliary, 92

 Valvular (cardiac) disease, chronic, 398
 treatment, 408
 Vaso-motor system, 315
 Vegetations, adenoid, 33
 Veins, diseases of, 460
 Venous circulation, 311
 hum, 358
 pulsation, 357
 Ventilation, 16
 Vital capacity, 12
 Vocal fremitus, 127

 White patches, 331
 "White pneumonia," 88

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